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INTRODUCTION

to

PLANT PATHOLOGY

BY

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INTRODUCTION TO PLANT PATHOLOGY

PREFACE TO THE SECOND EDITION

The rapid output of plant-disease literature since the publication of the first edition of the "Introduction" in 1937 is alone sufficient reason for the publication of a new edition. An attempt has been made to incorporate the new researches, especially those which bear directly on the types selected for detailed consideration. The amount of work required may be emphasized from a single illustration, apple scab, for which over 250 publications have appeared during the last ten years.

Certain additions and eliminations have been made on the basis of personal experience and the suggestions received from other plant pathologists. The sections on "Disease Prevention or Control" and on "Plant Pathology Methods" have been omitted, since the subject matter is generally covered in special courses following an introduction to the subject. Seventeen plant pathologists located in representative portions of the United States were asked to make suggestions, and their advice has been utilized in so far as possible. If all the suggestions received had been incorporated, it would have necessitated an expansion too great for a single volume. Fifteen additional types of parasitic diseases not treated in the first edition have been included, which has necessitated an increase in the number of illustrations from 200 to 240.

The author here wishes to express his gratitude for the various suggestions and for the friendly advice and criticisms that have been received from various pathologists in this and foreign countries. Special acknowledgment also is due to his associates in the Department of Plant Pathology for helpful suggestions and for material aid during the completion of this revision.

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PREFACE TO THE FIRST EDITION

This "Introduction to Plant Pathology" has been written as the result of a demand from teaching plant pathologists for a somewhat briefer treatment of the subject than is presented in the author's "Manual of Plant Diseases." This new work is not an abridgment of the more complete manual but involves much added material and an entirely different order of presentation. Experience has shown that many of the students in our agricultural colleges never complete any work in plant pathology beyond the elementary course. For this reason it has seemed advisable to include certain phases of the subject that were not emphasized in the editions of the larger work.

The new plan has involved an entire rewriting of the material presented and the omission of certain types discussed in the more complete work. The literature on plant diseases is rapidly expanding and an attempt has been made to incorporate the results of recent advances. References cited in the "Manual of Plant Diseases" are not duplicated, but only pertinent work which has appeared since the last edition in 1932. The pages of the "Manual of Plant Diseases" giving the earlier references are cited as (H) following the References.

An attempt has been made to give the student a general view of the relation of plant diseases to human affairs. Following the introductory chapters, the field of plant pathology as a whole is presented in the following sequence: parasitic diseases, including those caused by fungi, bacteria, seed plants and nematodes; virous diseases; nonparasitic diseases; plant-disease prevention or control; and methods of studying plant diseases. It is felt that this order of presentation will meet the needs of students and teachers better than the plan followed in the "Manual of Plant Diseases."

Many of the illustrations from the previous publication have been used and these are credited in the legends. The author is especially indebted to his coworkers, Dr. L. K. Jones, Dr. C. S. Holton and Dr. George Fischer, for suggestions and aid during the progress of the work. He also has profited by the suggestions and criticisms from the various plant pathologists who read portions of the original manuscript of the "Manual of Plant Diseases."

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    August, 1937.

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GENERAL INTRODUCTION

CHAPTER I

INTRODUCTION

Plant pathology is that phase of botanical science which deals with the *diseases* or *troubles* of plants. The knowledge concerning plant diseases now constitutes the field of *phytopathology* (Greek: *phyton*, plant; *pathos*, disease; *logos*, discourse), a science which has come to rank with horticulture, agronomy and soil science in the realm of plant industry. The *phytopathologists* are the trained plant doctors, the "medicine men of agriculture," whose final goal is successfully to prevent or control plant or crop diseases.

Disease Defined.—Plants may be considered from two points of view: (1) as individuals in plant society; (2) as producers of crops, or commercial products or as possessors of qualities desired by man. A slight modification of a general definition of disease should suffice to give a concept of disease as understood by the plant doctors. Disease may be defined as any alteration of the state of a plant or of some of its organs or parts, interrupting or disturbing the performance of vital functions, or as any departure from the state of health presenting marked symptoms or injurious effects.

Two types of disease may be recognized: (1) *systemic troubles*, or those which pervade or affect the entire plant; and (2) *localized diseases*, or those which affect only special organs or parts, such as roots, stems, leaves, flowers or fruits, or only certain portions of these organs. A few illustrations may serve to emphasize these types. A general yellowing of a plant indicating some impairment of the chlorophyll function, or a parasite or virus which pervades the entire plant or the major part of the plant causing a general derangement of certain physiological processes, is of the *systemic type*, while leaf spots, fruit spots or rots, localized killing of leaves, flowers, fruits, roots, bark areas, etc., are of the *localized type* and interfere only with the normal life processes carried out by the organs affected.

A more strictly agricultural aspect of plant disease is a "failure of thrifty development or a failure of a plant to produce a commercial prod-

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uct of satisfactory quality or quantity." The farmer is interested in both quality and quantity of production, and certainly one of the results of disease is the lowering of quality of our agricultural products, for example, scabby apples, smutty wheat, shriveled or shrunken grains, undersized fruits or root crops, etc.

Needs or Requirements for a Thrifty Development.—Since thrifty development has been emphasized in our concept of health, it is proper that the needs or requirements for a thrifty development should be briefly enumerated:

1. The proper inherent qualities of seed or stock from which plants are to be grown.

2. The proper environmental conditions of both air and soil—proper moisture, temperature and light relations; proper physical composition and aeration of the soil; and proper chemical composition of both soil and air in order that food materials may be available and toxic or poisonous substances be absent. For any given crop plant or variety of plant for each environmental factor there is an *optimum* which will induce the best development, while below the optimum and above the optimum the life processes will be slowed up until they either reach a very low ebb or cease entirely.

3. Freedom from mechanical injuries.

4. Freedom from infectious diseases, caused by plant parasites, animal parasites or viruses.

If any of these four factors or a group of these is seriously disturbed, disease in either mild or severe form will result.

Kinds of Plant Diseases.—According to the nature of the causal agent, three great groups of plant troubles may be recognized:

1. *Parasitic diseases*, including in the broadest sense all disturbances in the life and production of plants due to attacks by some other living organism, a *parasite*. The affected plant has long been designated as the *host*, but *suscept* has recently been used as a substitute. The parasitic diseases which fall within the field of plant pathology, are caused by: (*a*) *fungi*, of first importance, with many diseases for which they are responsible; (*b*) *bacteria*, second in importance to the fungi; (*c*) *seed plants*, furnishing a relatively small number of parasitic species, illustrated by the dodders, mistletoes, broom rapes and a few others; (*d*) the *algae*, including but a few parasitic species; and (*e*) *nematodes*, or round worms, including a small number of parasitic species, but of considerable importance. The consideration of all of the other depredations of animal parasites, including higher animals, insects, mollusks and protozoa fall within the province of either the *economic zoologist* or the *economic entomologist*.

2. *Virous diseases*, including a group of diseases in which the disturbed condition is the result of an infectious principle, a so-called "virus,"

which can be transmitted from diseased to healthy plants and can communicate the disease. The infective principle in the juice or cell sap of the diseased plant may be transmitted by mere contact, while with others, less infectious, juice inoculations are required, and, with the least infectious, budding or grafting is necessary. Most virous diseases are also transmitted under natural conditions by the feeding punctures of some insect visitor, a so-called "vector." Virous diseases are of the systemic type, the entire body of a sick plant being pervaded by the virus or the infective principle.

✓ 3. *Nonparasitic diseases,* including a great variety of nutritional disturbances due to lack of proper inherent qualities, to improper environmental conditions of soil or air, or to injurious mechanical influences.

The problems with which the professional plant doctor must deal call for the broadest kind of a training. He must be well grounded in the fundamentals of pure botany, including plant physiology, histology and taxonomy of seed plants; on account of the importance of fungous diseases, a detailed knowledge of systematic mycology is an essential part of his equipment; the increasing importance of bacteria as producers of plant disease would make him something of a bacteriologist; the similarity of plant troubles to those caused by insects leads him into the field of economic entomology; without a thorough training in chemistry and physics, he could make but little real progress in dealing with the nonparasitic diseases. Since the final goal of the plant doctor is the prevention of crop losses, or the destruction of plants or plant products, he must be in sympathy with agriculture in general and should have a good working knowledge of agronomic, soil and horticultural science.

The Beginnings of Plant Pathology.—Early historic and religious writings record the blighting, blasting, rusting or mildewing of the crops of the ancient peoples. Throughout the ancient era and the Dark Ages, but little was known of the true physiological behavior of plants; consequently, the ideas concerning plant diseases remained fragmentary and unorganized. Superstitions, dogmas and false interpretations of phenomena characterized the period. Even in the seventeenth and eighteenth centuries, there was but a slow dawning of our modern ideas of disease. The period from the early beginnings of civilization to 1853, with its slow advancement, may be called the "formative era of plant pathology."

The Early Modern Era.—This was ushered in by the publication, in 1853, of the classical work of Anton De Bary, the great German botanist and mycologist, on "Die Brand Pilze," in which he established the parasitism of the fungi associated with rusts and smuts. The causal relation of *Phytophthora infestans* to the late blight of potatoes was proved by his investigations published in 1861, and the establishment of the relation

of the aecidium on the barberry to the rust fungus on wheat followed in
1865. The brilliant mycological work of the Tulasne brothers and others
in France on the life history of ergot, powdery mildews, rusts and smuts
(1847–1863) and the contributions of M. J. Berkeley on British fungology
and vegetable pathology (1846–1860) are also landmarks in the early
part of the modern era. It remained, however, for Julius Kühn, a con-
temporary and countryman of De Bary, to produce the first modern
textbook on plant pathology—one based on the mycological discoveries
of his contemporaries. This work, entitled "Die Krankheiten der Kul-
turgewächse, ihre Ursachen und ihre Verhütung," appeared in 1858 and
presents such an accurate, concise and complete treatment of phyto-
pathology that Kühn may with justice be given the title of "father of
modern plant pathology." Soon after this, the "brilliant researches
and convincing demonstrations of that noted French savant, Louis
Pasteur, swept away the nebulous foundations of spontaneous generation"
(1860–1864), and improvement in culture methods soon followed which
made possible more accurate studies of the life histories of fungi and the
etiology of disease. /

The Place of Germany in the Early Modern Era.—The time was now
ripe for more rapid advances in plant pathology, important investigations
soon appeared, and manuals were published which were destined to mold
and direct the progress of plant pathological knowledge. A few of the
outstanding writers were Brefeld, Sorauer, Hartig, Frank, Kirchner, and
von Tubeuf. One of the most important works is the "Handbuch der
Pflanzenkrankheiten" begun by Sorauer in 1874 as a single volume but,
in its present edition (the fifth), expanded to six volumes with numerous
authors. While only prominent men responsible for manuals or texts
are here recorded, credit should be given to numerous investigators who,
by their careful and painstaking researches, placed Germany far in the
lead in the realm of plant pathology.

The Contributions of Other Foreign Countries.—While this develop-
ment of the science of plant pathology was proceeding in Germany, the
scientific workers in other countries were making important contributions
which were reflected in various manuals and texts. A few of the more
noteworthy authors may be recorded: Prillieux, Delacroix and Maublanc
in France, the first the author of a two-volume work on diseases of agri-
cultural plants (1895); Comes, Savastano, Berlese and Ferraris in Italy,
the latter the author of a two-volume manual (1913); J. Ritzema Bos in
Holland; Woronin in Russia; Rostrup in Denmark; Eriksson and Henning
in the Scandinavian countries; Ideta in Japan; Ward in England; and
McAlpine in Australia. This is but a brief survey of a few of the most
eminent phytopathologists of the countries mentioned who were respon-
sible for extensive treatises.

FIG. 1.—Four noted German plant pathologists. *A*, Julius Kühn; *B*, Anton De Bary; *C*, Robert Hartig; *D*, Paul Sorauer. (*From illustrations in "History of Plant Pathology," by H. H. Whetzel.*)

Phytopathology in America.—An account of the rise and development of plant pathology in America may now be presented. This subject was first taught incidentally with botany by Burrill in 1873 at the University of Illinois and as a special subject by Farlow in 1875 at Harvard. Two early events which gave impetus to the development of phytopathological investigations in the United States were: (1) the organization of a Section of Mycology in the Division of Botany of the U. S. Department of Agriculture (1885); and (2) the organization of the state agricultural experiment stations by the Hatch Act of 1887, which provided $15,000 annually to each state experiment station. This annual support for the state experiment stations has been supplemented by $15,000 from the

Fig. 2.—Class in plant pathology at the State College of Washington.

Adams Act of 1906, $60,000 from the Purnell Bill of 1925 and by the Jones-Bankhead Act of 1935 which provides additional support, the amount to be based on the rural population. Increased emphasis has been given to phytopathological investigations by additional support from these funds.

The Development of Plant Pathology in the U. S. Department of Agriculture.—With the gradual increase in financial support to the U. S. Department of Agriculture, the work in plant pathology has grown from its simple beginnings with its single worker to many divisions in the present Bureau of Plant Industry, with a whole army of plant doctors whose activities reach the remotest corners of the country. Plant-disease investigations in this bureau are now provided for in many different administrative groups, while in addition some plant pathological interests are provided for in other bureaus, the combined activities to

cover the investigational, extension and inspection, and control features of plant diseases.

The Development of Plant Pathology in the Various States.—The Hatch Act creating the agricultural experiment stations placed them with the agricultural colleges of the different states; consequently, these institutions have been the state centers of plant pathological activity, in many cases cooperating with the Federal departments. This direct support and encouragement to teachers and investigators from the land-grant colleges and by the Federal departments have made possible rapid strides in our knowledge of plant diseases. At first, teaching and investigation in plant pathology went hand in hand and were provided for as a part of the work of the departments of botany. In many states, teaching and investigation have now been separated, or separate departments of plant pathology organized. In most of the states the various lines of plant-disease work are provided for by:

1. Departments of botany or of plant pathology for teaching and for investigation of plant-disease problems, always at the state agricultural college and sometimes also in the separate State Universities.

2. Extension specialists in plant pathology in connection with the state extension service, varying from none to as many as five in some of the more progressive states.

3. State departments of agriculture in which the police or inspection duties relating to plant diseases are of first concern, and research and educational features secondary.

American Contributions.—In the early teaching of plant pathology in America, principal reliance was placed upon the German manuals and texts or English translations of these works. American workers have been so busy with experiment-station activities or investigations that they have had little time for the writing of books, with the result that many of the most noted pathologists of our country are known only from their published researches. The mills have been grinding steadily, and Federal and state bulletins and scientific periodicals are teeming with plant-disease information. The rapidity of our progress has militated against the production of textbooks, for the orthodox lecture of today is out-of-date tomorrow.

General manuals on plant diseases have been published by Freeman (1905), Duggar (1909), Stevens and Hall (1910 and 1921), Harshberger (1917), Heald (1926 and 1932), Owens (1928), Heald (1937), Melhus and Kent (1939) and Chester (1942).

Manuals dealing with specific phases or groups of plant diseases include bacterial diseases by Erwin F. Smith (1905, 1911, 1914 and 1920), diseases of tropical plants by Cook (1913), diseases of fruits by Hesler and Whetzel (1917), tree diseases by Rankin (1918), truck crops by

Taubenhaus (1918), and by Chupp (1925), citrus diseases by Fawcett and
Lee (1926 and 1935) and forest tree diseases by Hubert (1931) and by
Boyce (1938). Full titles of these publications and various others pub-

Fig. 3.—American authors of books on plant diseases. *A*, Charles Chupp, Cornell
University; *B*, Melville Thurston Cook, Insular Experiment Station, Rio Piedras, Porto
Rico; *C*, Benjamin Minge Duggar, University of Wisconsin, *D*, Edward Monroe Freeman,
University of Minnesota; *E*, John William Harshberger,[1] University of Pennsylvania;
F, Lexemuel Ray Hesler, University of Tennessee; *G*, Charles Elmer Owens, Oregon Agricul-
tural College; *H*, William Howard Rankin, New York State Department of Agriculture; *I*,
Erwin Frink Smith,[1] U. S. Department of Agriculture; *J*, Frank Lincoln Stevens,[1] Univer-
sity of Illinois; *K*, Jacob Joseph Taubenhaus,[1] Texas Agricultural Experiment Station;
L, Herbert Hice Whetzel, Cornell University. (*From photographs received from the several
authors.*)

lished in the English language will be found in the writer's "Manual of
Plant Diseases," Second Edition, 1932.

Landmarks of Plant Pathology.—A few of the outstanding discoveries
or events which have marked the progress of modern plant pathology
may now be outlined:

[1] Deceased.

Proof of the parasitism of fungi (rusts and smuts) by Anton De Bary, Germany (1853).

Proof of heteroecism of rusts, also by De Bary (1864).

Perfection of the plate method of isolating bacteria and fungi by Robert Koch (1881).

Proof of the relation of bacteria to the fire blight of the apple and pear, the first proved bacterial disease of plants (Thomas Burrill, University of Illinois, 1879–1881).

The discovery and introduction of our well-known Bordeaux for the control of downy mildew of the grape in France by Millardet (1882–1885), and its later adoption as the panacea for various plant diseases.

The discovery of the effectiveness of the hot-water treatment of cereals for control of smuts by Jensen of Denmark (1887).

The introduction of formaldehyde in the control of seed-borne diseases by Bolley of North Dakota (1897).

The demonstration of the effectiveness of lime-sulphur as an insecticide and fungicide and its use in the control of apple scab, by Cordley of Oregon and Piper of Washington (1906–1908).

The proof by Erwin F. Smith and C. O. Townsend of the U. S. Department of Agriculture of the bacterial origin (1907) of crown gall of various hosts and the assignment of the pathogene to a specific organism, *Pseudomonas tumefaciens.*

The organization of the American Phytopathological Society (1909) and the beginning of *Phytopathology* (1911) as the official organ of the society.

The enactment by Congress of the National Plant Quarantine Act (1912) and the creation of a Federal Horticultural Board to administer the act, aimed to exclude foreign insect pests and plant diseases.

The proof of the effectiveness of dusting with finely powdered fungicides and insecticides as a substitute for spraying, especially with finely ground sulphur and powdered lead arsenate (Cornell University, 1913–1917).

The first proof of the mosaic disease of tobacco as an infectious disease (1888) and the later rapid increase in our knowledge of the so-called virous diseases of plants, including mosaics, leaf roll and many other types.

The study of three important epiphytotics: (1) the *chestnut-tree blight,* a most virulent and devastating disease of chestnuts (1906 to present); (2) the *blister rust of the white pine,* a heteroecious rust, passing from currants and gooseberries to the white and other five-needle pines (mainly since 1915); and (3) *citrus canker,* a bacterial disease first introduced into Texas (1909–1910) and later spread to the other Gulf states, from which it has since been exterminated.

Emphasis on the production of immune or resistant varieties of plants for plant disease control, following the work of Orton on the Fusarium wilt of cotton, cowpeas and melons (1899–1909).

The campaign for the eradication of the barberry as a means of preventing the epiphytotics of stem rust of wheat, begun in 1917 as a war measure and continued to the present.

The introduction of the copper carbonate dust treatment of wheat for bunt or stinking smut (1921) and the more recent demonstration of the general application of other dust disinfectants for the control of seed-borne diseases.

The discovery of the function of the pycnia in rusts (Craige, 1927–1931) and demonstrations of the origin of physiologic strains by hybridization and mutation in the rusts and other parasitic fungi.

The conclusion that a virus is a highly infectious crystalline protein is based on the studies of Stanley and others (1935 and later).

CHAPTER II

SYMPTOMS OF DISEASE IN PLANTS

Every plant, in its particular way, shows when it is suffering from disease. Those concerned with the growing of plants should become familiar with those outward signs or symptoms which exist when plants are sick. In the nature of the resulting symptoms, there are no sharp and fast lines that can be drawn between parasitic and nonparasitic diseases, or between various "insect" troubles and those caused by bacteria or fungi. The first thing of importance to the grower is to detect

Fig. 4.—Powdery mildew (*Microsphæra alni*) of honeysuckle.

the presence of disease and then to learn its cause, the probable outcome and the need for or possibilities of control. The following brief survey of symptoms will be of service to the grower.

1. Discoloration or Change of Color from the Normal.—The most frequent discolorations are those affecting parts which are normally green, the presence of disease inducing a paling of the color which may finally reach a complete disappearance of the green pigment. Instead of showing the green color, the affected parts may be of a yellow cast, *etiolated* when the change is caused by insufficient light, or *chlorotic* when induced by other factors, while a complete absence of pigment results in *albinism.* A silvery sheen of normally green parts is also a characteristic accompaniment of some diseased conditions. Localized spots or areas showing whitish or gray, red or purple, brown, black, or variegated and

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FIG. 5.—Violet leaves affected with leaf spot (*Alternaria violæ*).   (*Photograph by F. D. Heald and F. A. Wolf.*)

FIG. 6.—Tar spots (*Rhytisma spp.*) of maple and willow.

concentrically zonate discolorations are signals of disease. The above manifestations of disease should not be confused with the common red coloration of early spring foliage or the autumnal changes preceding leaf fall.

2. Shot Hole or Perforation of Leaves.—The formation of localized lesions on leaves is frequently followed by the falling out of the dead tissue, leaving circular or irregular perforations which have suggested the term "shot hole." Certain species or varieties, especially of stone fruits, such as cherry, apricot or peach, are prone to develop the shot-hole symp-

Fig. 7.—Plum leaves showing an extreme case of shot hole due to *Coccomyces prunophoræ*.

tom. Shot-hole development may result from bacterial or fungous pathogens or from the operation of nonparasitic factors such as frost injury, localized action of toxic chemicals or nutritional disturbances.

3. Wilting.—The wilting of growing plants during a hot summer day followed by recovery is a normal occurrence and should not be confused with wilting as a symptom of disease. Two types of wilting from disease may be recognized: (*a*) the sudden falling over or "dropping dead" of young seedlings generally referred to as *damping-off;* and (*b*) the wilting without recovery of growing or adult plants. The juvenile wilting is caused by the invasion of a parasite in the young stem at or near the ground level, while in mature plants the water-conducting system ceases to function in the so-called "wilt" diseases.

4. Necrosis or Death of Parts.—Localized groups of cells in certain organs may die and assume a brown color as may be illustrated by the bitter pit or Baldwin spot of apples, or the internal brown spot of potato tubers. In other cases special organs of the plant such as leaves, stems or twigs, buds, flowers and developing fruits assume a dark or brown

Fig. 8.—*A*, internal brown spot of potato; *B*, bundle browning of potato.

coloration characteristic of dead plant tissue, producing a condition frequently referred to as "blight" of the affected organ.

5. Dwarfing or Atrophy.—Parts or organs such as leaves, fruits or flowers may be greatly reduced in size, suggesting such common names as "little leaf" disease of the apple or "little peach" with fruits of reduced size. In other cases the entire individual is reduced in size as a result of unfavorable factors, either environmental or parasitic.

6. Increase in Size or Hypertrophy.—Practically any plant organs, such as roots, leaves, fruits, etc., may be stimulated by the action of a parasite or by other factors resulting in enlarged and sometimes malformed structures. This hypertrophy may result from an increase in size of the component cells or from an increased cell division forming a larger number of component cells (hyperplasia).

7. Transformation of Organs or the Replacement of Organs by New Structures.—In the ergot of rye and other grasses (*Claviceps spp.*), certain of the ovaries are destroyed by the action of the parasite, and, in the place of the seeds, horny, elongated dark-purple spurlike bodies, the sclerotia or "ergots," appear. Stamens may become leafy in the green-ear or downy-mildew disease (*Sclerospora graminicola*) of *Pennisetum typhoideum* and other grasses; the whole staminate head, the tassel, or the ear, may become leafy in the head smut (*Sorosporium reilianum*) of corn; petals may become like sepals, stamens like carpels and carpels leaflike in the white rust (*Albugo candida*) of crucifers. The term "phyllody" is applied to the change of floral organs into leafy structures.

8. Mummification.—The transformation of fruits into shriveled structures called "mummies" is a phase of numerous diseases affecting our commercial fruits and may also occur in the fruits of wild plants. The fruit generally rots first, and during the change becomes filled with the mycelium of the parasite, after which it shrivels and becomes somewhat dry. Mummies may remain on the tree or fall to the ground, and, sooner or later, they give rise to a crop of spores which serve to spread the parasite. The formation of mummies is a very characteristic feature of the brown rot (*Sclerotinia spp.*) of stone or pome fruits. Other cases of mummification may be found in bitter rot and black rot of the apple and in the black rot of the grape.

9. Alteration in Habit and Symmetry.—Some plants which under normal conditions are more or less prostrate or creeping become ascending or even erect when attacked by a fungous parasite and, as it were, signal their distress. This is notably true in purslane (pusley), a common garden weed, when attacked by white rust and in certain spurges when harboring the cluster-cup stages of true rusts. In these and other cases a dorsiventral symmetry is changed to a more or less evident radial symmetry.

The vegetative organs may show various alterations; leaves may be changed from simple to irregularly lobed, from the ovate form to greatly elongated structures, or they may be variously twisted or deformed; stems may also be twisted or deformed, internodes elongated or shortened; and branching may be reduced or increased. Floral transformations are illustrated by the change of the club types of wheat heads to elongated ones by bunt or stinking smut, the flower head of an Acacia

to a spike by a rust infection, regular flowers to irregular, cyclic to strobilate types, dioecious to perfect and various other modifications.

10. The Destruction of Organs.—The complete destruction of organs may result either from nonparasitic causes or from the inroads of a parasite. Ovules may dry up without the formation of seeds from nonparasitic causes, or seeds may be destroyed by the action of a parasite as in smut of sheep sorrel or in the "bladder plums" due to *Taphrina pruni*. The so-called "seed" or fruits of cereals (the caryopses) are frequently destroyed by the operation of the parasites of the various kernel smuts (wheat, oats, sorghum), while in other cases the destruction is more complete and the entire inflorescence may be involved as in the loose smuts (wheat, oats, barley).

11. Dropping of Leaves, Blossoms, Fruits or Twigs.—This is, of course, to be considered as a symbol of disease only when it occurs prematurely or in excessive amount. Owing to a sudden change from a moist greenhouse to a dry room, from moderate to intense light or from cool conditions to warm, such house plants as fuchsias, foliage begonias, azaleas, rubber plants and many others may drop their leaves. The shedding of leaves from the action of parasites may be noted in the leaf casts of pine and larch, the leaf spot of alfalfa, and the leaf spot or yellows (*Coccomyces hiemalis*) of cherry. The shedding of blossoms may be illustrated by the nonparasitic blossom drop of the tomato or by the so-called "shelling" of grape blossoms or of partially developed berries. Nonparasitic dropping of fruits may be illustrated by the shedding of bolls in cotton or the June drop of stone or pome fruits, and shedding of fruits from the action of a parasite by severe infections of scab in apples or California blight in cherries.

12. The Production of Excrescences and Malformations.—The following abnormal formations may be included under this heading:

a. An abnormal development of hairs or trichomes from the surfaces of leaves giving feltlike patches, first believed to be of fungous origin and named *erineum,* but now known to be caused by parasitic mites and designated *erinose.* Erinose spots on grape are white at first, but dirty brown with age, those on the mountain maple bright red or scarlet.

b. Intumescences or pustulelike distentions of tissue, occurring most abundantly on leaves but also on stems or fruits, due to the abnormal elongation of groups of cells (Fig. 9). If the overdevelopment of cells is somewhat general, rather than distinctly localized, and extensive swellings of organs result, the condition is spoken of as "edema" or dropsy. Both of these abnormalities are of nonparasitic origin.

c. Galls, or localized enlargements on various organs in the form of small pustules or warts, larger tubercles, tumors or masses of cells making a morbid outgrowth of either fleshy or woody character, in which host

FIG. 9.—Leaf intumescence of *Cassia tomentosa*. (*After Sorauer.*)

FIG. 10.—Peridermium galls on *Pinus virginiana*.

tissues and parasites mingle. Some familiar illustrations in this group
are the small or large tumors of corn smut (*Ustilago zeae*), the so-called
"cedar apples" (*Gymnosporangium spp.*) of our common red cedar, the
elongated galls or "black knot" (*Dibotryon morbosum*) on twigs and
branches of cherry and "crown gall," the woody or fleshy outgrowths from
the crown and other portions of fruit trees and numerous other herbaceous
or woody plants.

d. *Cankers*, or localized lesions on woody or, more rarely, herbaceous
stems, which generally result in the corrosion and sloughing away of

Fig. 11.—Chestnut limbs showing cankers due to *Endothia parasitica*.

tissue with the final production of an open wound, exposing or penetrating
the wood. Cankers may be annual or perennial, that is, increasing in
size from season to season, and of either parasitic or nonparasitic origin.
Typical cankers are phases of the Pacific Coast and perennial cankers of
apple; the chestnut-tree blight or Endothia canker of American chestnut;
the European canker of apple and the fire blight of apple, pear and other
hosts, all of parasitic origin; while nonparasitic cankers are well illustrated
by "winter-sunscald" lesions which appear on the southwest side of tree
trunks or on the exposed faces of larger limbs.

e. *Witches'-brooms* or "hexenbesen," closely grouped clusters of fine
slender branches, generally arranged more or less parallel to each other,
and frequently originating from an enlarged axis. These may be illus-

trated by the witches'-broom (*Taphrina cerasi*) or the cherry, rust brooms on various coniferous trees and brooms on pine, larch and fir caused by scaly or dwarf mistletoes (*Razoumofskya spp.*).

FIG. 12. —Witches'-broom of service berry due to *Apiosporina collinsii*

f. Hairy root, or an abnormal number of fine fibrous roots, frequently making compact clusters. According to the location on the root or crown: *broom-root* type from a lateral root; *wooly knot* form with the roots from a crown-gall-like enlargement; and *aerial* with the roots or their primordia on the trunk or branches.

g. Rosettes, or closely grouped clusters of leaves caused by the failure of axes to make a normal elongation.   This should not be confused with the normal rosette habit of certain plants.   This habit characterizes certain diseases as the apple rosette of the Pacific Northwest, the pecan rosette of the southern United States and the rosette disease of peaches and plums in the southeastern states.

FIG. 13.—Fasciation of apple twigs.

h. The development of dormant or rudimentary structures, or of entirely new organs or structures, either similar to or entirely unlike any normal parts of the host.   Dormant buds may be started into growth stamens normally rudimentary may grow to full size, extra petals may appear, or an entirely dissimilar outgrowth may be formed as an out growth from the frond of a fern *(Taphrina laurencia* of Pteris).

i. Proliferation or prolification, that is, the formation of a new growth from an organ after it has reached the form or stage which would normally end its development.   Some typical illustrations are the formation shoot from the apex of a cone (larch), so-called

"sprouting pears" in which one pear forms directly above another or a leaf-bearing twig grows out from the calyx end; or a new rose from the center of an old one.

j. Rolling, crinkling or curling of leaves may result from either parasitic or nonparasitic factors. Leaf curl (*Taphrina deformans*), a fungous disease of peach; leaf roll, a virous disease of potato; and frost blistering and curling of apple leaves are typical illustrations.

k. Fasciation and spiralism are not uncommon types of malformation, the former resulting in a change of a cylindrical organ to a broad, flattened and more or less bandlike structure, the latter characterized by the change of a straight, cylindrical stem to form an irregular, scraggly structure with a flat or open spiral.

l. The abnormal roughening of surfaces, which under normal conditions should remain smooth. This may be illustrated by the russeting of fruits by frost or by sprays and by "scurf" or "scab" lesions caused by fungous parasites.

m. Fruits may be variously deformed, or root crops may be cracked, corroded or irregular in shape. Such changes may result from either parasitic or nonparasitic disturbances. Apples may be greatly reduced in size and malformed by the scab fungus or by freezing when the fruits were very young, but it should be kept in mind that insect pests also may be the cause of deformities.

13. The production of an exudate from certain plant organs is an accompaniment of certain diseased conditions, but should not be confused with the normal physiological processes of *guttation* or the forcing out of cell sap on a free surface, or with bleeding from cut ends of stems or other plant organs. Exudates may be classified according to their causes and their composition as: (*a*) *bacterial* exudates, well illustrated by ooze from lesions of fire blight on apple, pear and other hosts; (*b*) *slime flux*, a semifluid or fluid outflow from the bark or wood of various deciduous trees; (*c*) *gummosis*, resulting in formation of clear or amber-colored exudates which set into solid masses upon the surface of affected parts, common in stone fruits and citrus species; (*d*) *resinosis*, or the abnormal exudation of resin or pitch from coniferous species; (*e*) *latexosis*, or an abnormal outflow of latex, a milky fluid characteristic of certain species.

14. Rotting of Tissue.—Succulent or woody stems and roots, fleshy leaves, flower buds or fruits may be affected with either dry or soft rot— the "gangrene" of plant tissue. The character of the rot may depend on the structures involved, the causal factors or complications and external conditions, but two types are recognized: soft or watery rots and dry rots. Rots may be grouped according to the special structures or organs involved as: (*a*) *root rots*, illustrated by decay of such root crops as beets,

carrots, parsnips, turnips, sweet potatoes, etc., the invasion of fleshy roots of such herbaceous crops as cotton or alfalfa and the destruction of woody roots of our forest, shade or fruit trees, and shrubs; (*b*) *leaf or stem rots*, illustrated by the decay of succulent or herbaceous leaves or stems as in the late blight of potatoes, the slimy soft rot of lettuce or the stem rot or wilt of various garden and field crops; by the decay of modified stems such as tubers, rhizomes, bulbs or corms; and by the decomposition of the woody stems including live standing timber, dead timber and timber

Fig. 14.—Cross section of the fruit of an eggplant rotted by *Phomopsis vexans*.

products such as posts, poles and lumber; (*c*) *bud rots* or the decay of fleshy buds of which the bud rot of carnations, the bud rot of the coconut and the black rot of the cabbage are notable examples; and (*d*) *fruit rots*, which are soft or dry, varying with the nature of the fruit, the causal agent, and the environmental factors which are operative.

The great majority of rots are of parasitic origin and are induced by either bacteria or fungi. Rotting may, however, result from the operation of nonparasitic factors, as in the so-called blossom-end rot of the tomato, or the blossom-end rot of the watermelon. Rots of various kinds are the cause of heavy losses to growing crops or standing timber, and to vegetables or fruits while in storage or during transportation to market.

CHAPTER III

THE RELATION OF FUNGI AND BACTERIA TO HUMAN AFFAIRS

In a study of the diseases of plants much attention must be given to the importance of fungi and bacteria as causal agents. Lest this emphasis might leave a false impression as to the other activities of these organisms, it may be well to outline briefly some of the useful relations of fungi and bacteria in the world of living organisms and also some of the other harmful relations besides the production of diseases. The fungi and bacteria which cause disease are "robbers" which steal their food from other living organisms, but there are many other species of fungi and bacteria which live upon the dead remains of animals and plants. These lowly organisms are playing a very important role in nature.

Fungi and Bacteria as Scavengers.—Green plants by means of their chlorophyll apparatus and the energy of sunlight are using the carbon dioxide of the air as the source of their carbon food supply and are building it into their structure. The supply of this gas in the atmosphere is relatively small—only about 0.25 per cent. Unless this supply is renewed, it would soon all be locked up by this continued use. This supply of carbon dioxide is renewed in four ways: (1) through the breathing or respiratory processes of plants, animals and mankind; (2) by the burning of plant structures or of coal; (3) by the expulsion of this and other gases from volcanoes; and (4) by the decomposition of plant and animal remains. The first three are of minor importance in replenishing the supply of carbon dioxide as compared to the last or the constant breaking down of the vegetative cover following the death of annual plants, the fall of leaves, the windfall of limbs and entire trees. If it were not for the activities of these fungous and bacterial scavengers, the surface of the earth would become clogged with the accumulating remains of green plants, and, in the course of a few years, the world supply of carbon dioxide would be exhausted. In these processes of rot or decay by which plant remains soften, crumble and become a part of the surface soil, carbon dioxide is constantly being returned to the air and the never-ending cycle is continued.

USEFUL RELATIONS OF FUNGI

Fungi as Food.—Many of the higher fungi that grow in the fields and woods are edible while others are either poisonous or are lacking in

desirable qualities. According to the popular impression, the edible forms are classed as mushrooms and the poisonous forms as toadstools. Any of the conspicuous, fleshy fruits of the higher fungi whether Ascomycetes or Basidiomycetes, may be classed as mushrooms or as toadstools, without any regard to their edible or poisonous properties. The two terms are used interchangeably, but it is more common to apply the name toadstool to the gill forms represented by the cultivated mushrooms. Mushrooms may be used fresh or they may be dried or canned alone or used as important constituents of soups, sauces, catchups, etc.

A brief outline of the groups and genera furnishing edible species may be presented:

1. *Ascomycetes or sac fungi:* the saddle fungi represented by species of Helvella and Gyromitra; the morels or species of Morchella and Verpa; the truffles including several species of Tuber; Cyttaria species, parasitic on the evergreen beeches in Chile, Patagonia, Tasmania and New Zealand; and several species of lichens, notably *Cetraria islandica*, of northern latitudes, used as an article of diet for man and domestic animals; *Cladonia rangiferina* or reindeer moss, an important source of forage for the reindeer of northern regions, and *Lecanora esculenta* of the steppes of southwestern Asia and southeastern Europe. This lichen forms thick, wrinkled crusts on stones, preferably on small fragments of limestone; when older they separate from the stones, roll up to form spherical, warted bodies, from the size of a pea to that of a hazelnut and when loose they may be rolled away by the wind. After storms they may be piled up behind bushes or washed into depressions by rain water and can be collected in quantity. This lichen is the manna sent to the Israelites on the journey out of Egypt into the Holy Land.

2. *Basidiomycetes or basidium fungi:* the Jew's-ear, *Hirneola auriculajudae* and *H. polytricha*, the latter used by the natives of China, New Zealand and the South Sea Islands; many species of the gill fungi which include the common cultivated species of mushrooms, *Psalliota (Agaricus) campestris* and *P. arvensis;* some species of pore fungi from the genera Boletus, Strobilomyces and Fistulina, characterized by some popular writers as "neglected beefsteaks"; some species of the tooth fungi or Hydnaceae, especially *Hydnum coralloides*, *H. caput-medusae* and *H. erinaceus*, the latter sometimes called the hedgehog fungus; certain species of the club fungi or Clavariaceae, especially *Sparassis crispa* and numerous species of Clavaria, some of which have been likened to noodles and macaroni and may be used in the same way; false truffles represented by certain species of Rhizopogon, especially in Japan, by *Scleroderma vulgare* and by the "red truffle," *Melanogaster variegatus;* also the giant puffball, *Calvatia maxima* and numerous species of puffballs of the genus Lycoperdon.

In only a few cases are fungi cultivated in the same sense as other crops but sometimes special procedures are invoked to stimulate their production under natural conditions. The following may be noted: (1) *Truffles.* The truffle industry is carried on chiefly in southern France and the commercial product is obtained in two ways: (a) by "hunting" the truffles in their natural habitat, using trained dogs (sometimes pigs) which locate the subterranean fruits buried at a depth of 2 to 8 inches and generally associated with oaks, beech and hazelnut trees; and (b) by cultivation. In one method a natural habitat is selected, enclosed and sown with acorns; after several years growth of the saplings, truffles appear and may be collected for many years. In a second method a suitable location is selected, the trees thinned, and truffles brought from their natural habitat are planted in trenches. This method is used extensively in oak plantations in southern France. (2) *The cultivation of the Jew's-ear in China.* This is *Hirneola polytricha,* a fungus which grows naturally on decaying oak stumps under moist conditions. In the artificial production, small sapling oaks are cut down, the branches removed and the trunks cut into poles 8 to 10 feet long. These are left scattered on the ground and in several months become infected, after which they are stacked slantingly in groups. The brown fruits develop the following year and are collected for market, and are a regular export article from Manchuria. (3) *The cultivated mushrooms.* The cultivation of *Psalliota campestris,* probably began in France near Paris, has been extensively developed in that region and more recently in England, the United States and other countries. In all of these countries use has been made of stone quarries, in some cases of extensive mine-like tunnels below the surface; special sheds of lumber or mushroom houses have been used extensively in the various countries, especially America, and, in some localities, beds have been prepared under glass or even in the open. A description of the details of preparation of the beds from stable manure and soil, planting the beds with the mushroom spawn, and casing the beds with loamy soil is beyond the scope of the present discussion. Brief mention may be made of the cultivation of morels which, to a small extent, are cultivated in France using methods similar to those used with the common mushroom, but planting the beds with cut pieces of the fructifications; also of the extensive "shii-take" industry of Japan. The agaric, *Armillaria shii-take,* is grown from the logs of oaks and some other species. The logs are cut, soaked in water for some days, and pounded to loosen the bark; holes a few inches deep are bored in them at intervals, inoculated with pieces of wood containing the mycelium, and the logs left in a shaded part of the forest. The first crop is harvested after about two years, and, by proper rotation, mature mushrooms are available at different seasons or in the spring, summer and autumn.

Fungi as Drugs.—Parallel with the general decline in the use of medicinal herbs, fungi have come to have a very restricted application in modern Materia medica. In early times *Polyporus (Fomes) officinalis* under the name of "agarick" was a universal medicine; certain forms had a cathartic effect, some were recommended as styptics. Many other specific applications of fungi were favored in medieval and later times but all except a very few of these disappeared. Those which appear in modern pharmacopoeias are ergot or *Claviceps purpurea*, corn smut or *Ustilago maydis*, Iceland moss or *Cetraria islandica*, the purging agaric or *Polyporus officinalis* and yeast or *Saccharomyces cerevisiae*. Ergot is the only one of the list recognized as official in American and British pharmacopoeias.

Industrial Applications.—The most important industrial employment of fungi is the use of yeasts and a few other fungi because of their ability to induce the alcoholic fermentation. The common yeast *Saccharomyces cerevisiae*, used in the baking of bread, is generally available in either the dried yeast cakes or in the form of compressed yeast, the latter being more extensively employed. In the raising of bread, the yeast, under the influence of moisture, heat and food supply, begins to multiply and ferments some of the sugar contained in the dough with the production of carbon dioxide and alcohol. The production of gas bubbles in the sticky dough causes it to swell or to become light or to "raise." The heat of baking causes the evaporation of the small amount of alcohol produced.

Yeasts find very extensive use in the manufacture of alcoholic beverages from the fermentation of fruit juices: wines from the grape, cider from the apple, perry from the pear, and wines from other fruits such as raspberry, blackberry, loganberry, elderberry, etc. The wine yeasts have been generally included in the composite species *Saccharomyces ellipsoideus*. Malt liquors or beer, porter, lager and ale are prepared from the malt of barley (sometimes other grain), which is subjected to the process of mashing, and the beerwort, so formed, is fermented in vats by the addition of some one of the pure-culture strains of *S. cerevisiae*. After some days when the fermentation has subsided and the sugars and dextrins have been converted into carbon dioxide and alcohol, the beverage is placed in large casks for aging which requires a few weeks to several months. Among other fermented beverages are the following: *kumiss* prepared in Russia from mare's milk; *kefir* produced by the natives of the Caucasus by fermenting milk with "kefir grains"; *palm wine* from the juice of palms obtained by cutting off the terminal bud; the Mexican drink, *pulque*, from the juice of the century plant or Agave; *ginger beer* by fermenting a mixture of ginger, sugar and water with the "ginger-beer" plant; *pombe*, an African drink prepared by fermenting of malt made from millet seed; and *mead* made by the fermentation of honey

after the addition of water and certain salts. Various yeasts assisted by bacteria are active in the preparation of these beverages. Several species of filamentous fungi play a part in the production of alcoholic beverages. *Mucor oryzae* and *Mucor rouxii* are constituents of Chinese and Javanese raggee used in the manufacture of *aarack*, while *Aspergillus oryzae* is used in the saccharification of rice starch in preparation of the Japanese drink *sake*.

Beverages of several types are obtained by the distillation of yeast-fermented juices; *brandy* from fruit juices; *rum* from fermented molasses; *whisky* from a fermented mash prepared from malted grain. The fungi mentioned above may be utilized in one of the methods for the saccharification of starch in the preparation of commercial alcohol by distillation, but heating with acid and the malting of grain are more generally employed.

Certain fungi play a prominent part in giving character to some varieties of cheese. Camembert cheese, a soft type, owes its specific character to *Penicillium camemberti*. which secretes proteolytic enzymes. Roquefort cheese, originally prepared from goat's milk but now made also from cow's milk, owes its specific character to another mold, *Penicillium roqueforti*. The cheese is inoculated with cultures of this organism, and its growth through the cheese is made possible by perforations which admit the air. It is also claimed that gammelost, a variety of Norwegian cheese, owes its special flavor to *Mucor casei* and *Chlamydomucor casei* which degrade the casein and also form pungent fission products.

Mention should also be made of the use of lichens in furnishing certain reagents or dyes: for example, litmus from certain species, especially *Roccella tinctoria*, *Lecanora tartarica* and others; dyes, such as archil (orchil) and cudbear from the above species and also from others. The so-called "green oak" used in the manufacture of Tunbridge ware is common oak which has been colored by the growth of the fungus, *Chlorosplenium aeruginosum*, but this color is now much imitated by artificial staining. Certain polypores, especially *Fomes fomentarius* were formerly much used in the manufacture of amadon or German tinder. Various species of fungi have found domestic employment (rarely commercial) as a source of dyes, snuff, ink, bottle corks, fly poison, etc.

Mycorhiza.—A nonpathogenic relation is established between certain fungi and vascular plants and also between fungi and some bryophytes. Since in most cases the associations are between fungi and the roots of seed plants this living together or a symbiosis has been named mycorhiza. Only brief mention can be made of these interesting relations which are of two general types: (1) *ectotrophic* or a mantle of mycelium about the tips and younger portions of the roots, with more or less internal penetration; and (2) *endotropic* or with a more or less exten-

sive distribution of intracellular mycelium with a variable development of hyphae on the surface or outside of the root. Two general views have been held: (1) that these root fungi are harmless parasites; (2) that the association is beneficial to one or both of the symbionts. Modern investigation, especially with forest trees, orchids and heaths, point to the truth of the second view, especially that the association is of benefit to the vascular hosts. The great majority of the mycorhizal fungi appear to be Hymenomycetes which are present in the humus-containing soils of the environments in which their vascular associates are flourishing.

Parasites of Insects.—A large number of fungi are parasitic on insects and in the majority of cases the insects that are parasitized are the enemies of crop production. In certain cases these parasites help in reducing the prevalence of injurious insects under natural conditions, and, in some cases, their natural distribution has been supplemented by spraying the trees with water containing spores from cultures. Some of the more important insect-attacking fungi are species of Empusa and Entomophthora attacking flies, aphids, grasshoppers and caterpillars, Cordyceps species which kill certain caterpillars and beetle larvae, and representatives of various genera which parasitize scale insects and white flies of citrus in humid localities of Florida, the West Indies and other countries.

HARMFUL RELATIONS OF FUNGI

In other chapters attention will be given to the numerous diseases of growing plants caused by fungi and also to losses of fruits and vegetables while being held in storage, on the market or in transit to market. Some of the other harmful relations follow.

The Decomposition and Staining of Lumber and Wood Products.—Various fungi may cause a decomposition or decay of lumber or timbers when in contact with moisture, or others may be responsible for staining of lumber and wood under varying conditions of usage and exposure. Brief consideration may be given to the injury or damage caused by the action of saprophytic fungi on wood.

1. *Dry Rot in Buildings.*—Dry rot in the foundations and walls of buildings is caused by a number of fungi, especially *Coniophora cerebella* responsible for a cubical brown rot, the common dry-rot fungus, *Merulius lachrymans* and other species of Merulius; also by the building poria, *Poria incrassata* and representatives of other genera. Dry rot in buildings is a factor of importance in all countries, but is the most serious in regions of abundant moisture and where temperature ranges of 68 to 97° are common. If the wood is dry or contains very little water (20 per cent) it will not decay except under special conditions and also if completely saturated with water no decay will result. Dry rot may occur in build-

ings which are generally kept dry, such as private residences, factories or mills, but is more likely to be serious in buildings in which the humidity is high, such as canning factories, creameries, pulp mills, etc. Dry rot is most serious in the United States in the southern humid areas and in the humid coast country of the Pacific Northwest. The dry-rot fungus *M. lachrymans* is especially destructive since, after it is once established in the foundation timbers of a building, it may continue to grow and disintegrate dry wood through its ability to liberate moisture by the decompositions which it initiates.

2. *Decay of Railroad Ties, Posts, Poles, Lumber, etc.*—Railroad ties, fence posts, telephone poles, piling and mine timbers are generally exposed to conditions favorable to some development of wood-destroying fungi during storage; when placed in use, they are in contact with soil moisture which will favor the growth of any decay fungi already present in the wood and will offer the opportunity for new invasions. The length of life of ties, posts and poles is greatly increased by the use of waterproofing, or by the impregnation of the wood under pressure by chemicals which retard the development of the decay fungi. Such protective treatments are now extensively used by railroad, telegraph and telephone companies. The fungi which cause the decay are of two types: (*a*) those which attack the living trees and continue to develop after the trees are cut and (*b*) those which are new invaders, either during storage or after the various products have been put in use. Various gill and pore fungi of the following genera are among the most important agents of decay of ties, posts, poles and mine timber: *Pholiota adiposa*, the yellow-cap fungus and *Lentinus lepideus*, or scaly-cap fungus and *Armillaria mellea*, the honey mushroom (gill fungi); and species of Glœoporus, Polyporus, Polystictus, Daedalia, Fomes, Trametes and Lenzites (pore fungi). Logs held in storage for various purposes or bolts for use in paper manufacture suffer deterioration from many of the same fungi already noted which cause decay and from others which invade and discolor the wood. Lumber held in stacks in mill yards, especially in southern regions, also may suffer heavy deterioration from the various wood-rotting fungi. Wood pulp must frequently be stored previous to manufacture into paper, and it also suffers from wood-rotting and staining fungi.

3. *Staining of Wood.*—Lumber and any raw wood products exposed to the weather for any considerable period may be stained by the growth of fungi, which are generally not decay-producing forms, and thus be lowered in grade or value; also chemical stains may occur but these are of minor consequence. Packing boxes, baskets, etc., used in handling fruits and vegetables become discolored by the combination of plant juices and various molds, the former furnishing added food material for the nutrition of the contaminating fungi. Most of the sap-stain fungi

are Ascomycetes and imperfect fungi and may cause injury in two different ways: (a) by a superficial growth which does not penetrate the wood and cause interior discolorations; and (b) by an internal penetration with the accompaniment of discolorations below the surface. Besides the fungi causing blue stains, there are other colors such as yellow, red, green, olive and nearly black. The annual loss in the United States alone from sap stains is estimated at $10\frac{1}{2}$ million dollars.

The Molding of Foodstuffs.—The spoilage of cooked, canned, preserved or processed foodstuffs, including both plant and meat products, from the development of fungi is a matter of both household and packing house experience. Bread and cake, if held too long, may become moldy, jellies and jams may develop a surface growth of mold unless carefully protected, home-canned fruits if insufficiently sterilized or imperfectly sealed may be spoiled by the growth of yeasts and molds, and even commercial catchups are frequently ruined or badly contaminated with molds. Even many of the commercial brands of maple sirup will develop a surface growth of mold if left exposed to the air. Canned, sweetened condensed milk may contain masses of mold known as "buttons." Meat and meat products such as hams, sausages and bacon may support molds which reduce their value. Many different filamentous fungi and some yeasts are found on the various products. Species of Penicillium and Aspergillus are perhaps the most frequent, but many other genera are represented, including Mucor, Rhizopus, Oidium, Monilia, Monascus, Alternaria and Fusarium.

Molding of Cheese and Butter.—It has been pointed out that certain types of cheese owe their character to the presence of specific fungi, but others may develop abnormal growths either upon the surface or upon the exposed surfaces when cut. Common cheese will mold even when held at icebox temperatures, generally from the presence of certain Penicillia. Butter is subject also to mold spoilage, three different types of abnormalities being recognized: dark or smoky spots or areas caused by forms like Alternaria and Cladosporium with dark mycelium and dark spores; green patches or spots caused by Penicillia; and patches of yellow or orange produced by *Oidium lactis*.

The Molding or Mustiness of Animal Feeds.—Wheat or any of the other cereals, if exposed to humid conditions too long in the field at harvesttime or if not sufficiently dry when threshed and stored, may deteriorate from the growth of various fungi and develop a condition generally characterized as *mustiness*. This condition may also be imparted to ground feeds or meal and may be aggravated still further during storage. Cereal or grass hays that are not properly cured may develop a similar musty condition. Many forms of saprophytic fungi, including species of Penicillium, Cladosporium, Alternaria, Aspergillus,

Mucor, Fusarium and Dematium are commonly prevalent and contribute to the mustiness. A red coloration of silage is caused by the primitive ascomycete, *Monascus purpureus.*

The Molding of Leather and Fabrics.—Defects in leather may be caused by molds which develop after tanning and before final coloring. Manufactured leather articles, if exposed to humid conditions, may develop conspicuous coatings of blue or other colored molds, especially species of Penicillium and Aspergillus. Wool and woolen fabrics may also be attacked by various fungi and also some bacteria; much attention has been given to chemical treatments for the reduction of these injuries. Cotton fabrics or other cotton articles, especially those containing sizing may suffer much deterioration from the work of various fungi. The conspicuous mildewing of tarpaulins, tents, cordage, fishing nets, etc., is due to the growth of various fungi.

Fungous Diseases of Various Forms of Animal Life.—Fungous para-sites of injurious insects have already been discussed under useful relations of fungi. The present interest is with fungous diseases of economic forms of animal life and the influence of such effects on man. Fishes and other aquatic forms of animal life are attacked by various water molds or Saprolegniaceae. Goldfish and other fish kept in aquaria are frequently attacked; serious epidemics among salmon have been recorded, one of the most destructive parasites being *Saprolegnia ferax.* The silkworm and the honeybee, two of the most useful insects are affected by serious fungous diseases; the most serious for the former is calcino or the "muscardine" disease caused by *Botrytis bassiana,* sometimes very prevalent in France; and the brood and adult disease of the honeybee caused by *Aspergillus flavus.* Many different fungi have been described as associated with diseased conditions in man and animals and in certain cases are known to be definitely pathogenic. Fungous infections of man and animals are generally referred to as mycoses. The lungs, throat, ear, skin and the hair are the main parts of the body invaded, more rarely internal organs. Pathogenic forms of Mucor and Rhizopus are known in both man and cattle. Sporotrichum species are very important pathogenic forms, as many as 13 different species having been described as causing sporotrichosis. *A. fumigatus* is the cause of lung infection in domesticated birds, pigeons, chicken, ducks and turkeys; lesions may also be formed in the lungs of cattle, sheep and horses, and in man ear infections occur frequently, but lung infections only rarely. The der-matophytes are those forms producing lesions either on the scalp or the smooth skin, and include species of Achorion, Microsporum, Trichophy-ton, Epidermophyton and Endodermophyton. *Achorion schoenleinii* is the cause of favus and various types of ringworms by the other genera. The infections of feet and hands common in athletes are caused mostly

by species of Trichophyton. Among the other fungous infections are blastomycosis caused by *Oidium* (Blastomyces) *dermatitidis;* another closely related disease, coccidioidal granuloma; thrush caused by *Monilia albicans* or similar forms; sprue, a disease of the tropics believed by some to be caused by a Monilia; and certain yeast infections of the skin or of the brain and meninges.

Three fairly well-defined types of infection in man and domestic animals are caused by actinomycetes: lumpy jaw caused by *Actinomyces bovis;* Madura foot caused most commonly by *A. madurae;* and a third or acid-fast group represented by *A. asteroides* in man and *A. farcinica* in cattle. It is believed that the infection in cattle is contracted from contaminated hay, straw or grain and in man from other contaminated vegetable matter. The lesions occur most frequently in the mouth parts, but primary lesions may develop on other parts of the body, even the lungs or the intestinal tract.

Toxic Effects on Man and Animals.—Much has been written about the possible toxic effect of various moldlike fungi on food to man and on forage or feeds to animals, but in general there is little evidence that these forms are toxic when entering the digestive tract. Some forms have shown toxicity when injected directly into the blood stream. The toxicity of ergot to man and animals is discussed under the treatment of this disease of rye and other cereals.

Many species of the higher fungi represented by certain mushrooms contain highly toxic substances. Four different groups of mushrooms may be recognized according to their toxic effects: (1) Toxic after a period of incubation, and causing degeneration of nerve and gland tissue. This group is represented by *Amanita phalloides,* *A. virosa* and *A. verna.* It is claimed that these three species are responsible for 90 per cent of the deaths from fungous poisoning. (2) Causes gastric disturbances and excites and then paralyzes the central nervous system. The best-known representative of this group is the fly agaric, *Amanita muscaria.* Although this fungus may be deadly it has been employed in certain portions of Russia as a stimulant because of an effect similar to hashish. (3) Produces gastroenteritis by direct action on the mucous membrane of the digestive system. It is rarely lethal but causes pains, vertigo, vomiting and diarrhoea. This includes the acrid species of Russula, Lactarius, Boletus and some others. (4) Species containing hemolytic or blood-destroying principles. This group is represented by Morchella, Helvella and Gyromitra species, reputed edible forms containing unstable *hemolysins* which are destroyed by cooking. •

USEFUL RELATIONS OF BACTERIA

The most important useful relations of bacteria are as follows: in connection with the transformations of organic and inorganic material

in the soil; as the most important agents in the fixation of atmospheric nitrogen; for the part which they play in dairy manufacturing; for their utilization in various industrial processes; and in the preparation of certain agricultural products.

Transformations of Organic and Inorganic Materials.—The part which bacteria and fungi play in the putrefaction and decay of plant and animal remains has already been emphasized. There are three important steps in the transformation of humus nitrogen in the soil: (1) the production of ammonia; (2) the change of ammonia to nitrites; and (3) the transformation of nitrites to nitrates. The bacteria which decompose protein compounds with the production of ammonia are called "ammonifying bacteria," and many different kinds exist in soils. Following the production of ammonia, other bacteria, called "nitrate bacteria," transform the ammonia into nitrites (NO_2), while still others transform the nitrites into nitrates (NO_3) which are then available for the use of our crop plants as their source of nitrogen. The changes induced by the nitrite and nitrate bacteria are included under the more restricted use of the term "nitrification," a process of great practical significance to agriculture. Under certain conditions the reverse process of denitrification breaks down nitrates and nitrites to form ammonia compounds which may be lost to the soil.

While the decomposition of the cellulose of plant residues is carried out in large part by the digestive action of fungi, certain cellulose-fermenting bacteria are also active. These changes are occurring where there are accumulations of vegetable matter exposed to suitable conditions of moisture and temperature.

Nitrogen Fixation.—Owing to the various ways in which nitrogen may be lost from the soil, the world supply of this essential plant nutrient would soon be reduced if it were not for the ability of certain bacteria and crop plants to draw directly upon the supply of free atmospheric nitrogen. This utilization of free atmospheric nitrogen is known as *nitrogen fixation* and is accomplished by (1) free-living or nonsymbiotic forms of bacteria which are independent of crop plants; and (2) symbiotic bacteria, the most important forms of which may live for a time free but become associated with the roots of leguminous plants in root tubercles or nodules.

The free-living forms of nitrogen fixers are of two types, anaerobes and aerobes. The anaerobic forms may be illustrated by *Clostridium pasteurianum*, capable of growing along with other forms which use up the air. The aerobic forms are more important and more numerous and include six or more different species of Azotobacter, with varying capacity to fix nitrogen. Their power to fix atmospheric nitrogen is greatest in soils deficient in combined nitrogen and sinks to either nil or to a low level in rich soils. The aerobic forms are widely distributed in arable

soils and under proper conditions of cultivation of soils for summer fallow and for certain crops may greatly increase the nitrogen content of the soil. It has been claimed that nitrogen has been increased by nitrogen fixation under some conditions to such an extent as to cause injury to crops (see Niter Burning).

The symbiotic nitrogen-fixing bacteria are best illustrated by the forms associated with the root tubercles or nodules of various legumes such as clovers, alfalfa, beans, etc. This association is necessary for the proper development of leguminous crops and is responsible for the value of legumes as enrichers of the soil in contrast to nonleguminous crops. Originally the legume bacteria were supposed to represent a single species *Rhizobium leguminosarum*, but now several species and strains are recognized (Rhizobium, Bacterium and Pseudomonas are the generic names used by different authors). The legume bacteria may be present in many cultivated soils or if absent may be introduced by inoculation of either seed or soil.

The entrance and establishment of the nodule bacteria is brought about in the following way: (1) by penetration of and development in a root hair and the formation of a zoogloeal strand which advances into the root cells; (2) the development of a nodule with the central cells well filled with the bacterial rods; and (3) the transformation of the rod-shaped bacteria into irregular, enlarged forms known as "bacteroids."

By cross inoculations using pure cultures, it has been shown that different species or possibly physiologic strains exist which are able to associate themselves only with particular species of legumes. At least seven different groups of the bacteria have been established for cultivated legumes, which may be designated by one of the crop plants with which they are associated, as follows: alfalfa, red clover, vetch, garden and navy bean, lupine, cowpea and soybean. In addition other strains have been cultured from wild species of legumes. At the present time various experiment stations and private companies are furnishing pure cultures of the various strains, which may be used by mixing with the seed previous to sowing.

Iron and Sulphur Bacteria.—The iron bacteria are represented by certain species of Cladothrix, Crenothrix and Leptothrix, genera of higher or filamentous bacteria. It is believed that these forms have played an important role in the past and are also at the present active in effecting the accumulation of iron compounds. Through the agency of these iron bacteria, deposits of bog iron ore have been formed and are being formed in meadows, marshes and swamps. Sulphuretted hydrogen is one of the products evolved in the decomposition of protein when organic debris is broken down by bacteria. This is not available to higher plants as a source of sulphur, but must be changed into sulphate before

it can be used. This change can be accomplished by certain of the bacteria known as sulphur bacteria. These bacteria are especially abundant in locations where there is a more or less constant supply of sulphuretted hydrogen, and they appear to be dependent on this compound for their energy.

The Use of Bacteria in Industrial Processes.—Vinegar, which is a solution containing acetic acid, is produced by the fermentation of certain alcoholic solutions by means of acetic-acid bacteria. Cider, grape juice or other fruit juices or honey or sugar solution may be used. The first step is the alcoholic fermentation induced by yeasts followed by the oxidation of the alcohol to acetic acid by the enzymes produced by the acetic-acid bacteria. The growth known as "mother of vinegar" that forms on the surface of exposed alcoholic solutions is composed in large part of acetic-acid bacteria. Vinegar may be made in the home by allowing any of the sugar-containing juices to ferment first to alcohol and then to acetic acid. This is a slow process but may be speeded up somewhat by the addition of some mother of vinegar. In the commercial manufacture of vinegar, various methods have been adopted for speeding up the process, the most successful being the so-called "quick" or German method.

In the process of *retting* of flax or hemp, bundles of these plants are soaked in water. Bacteria which develop produce pectinase, an enzyme which dissolves the middle lamellae holding the fibers together and thus brings about their separation. Following the retting, the bundles are removed from the water, they are dried and the fibers are separated by mechanical means. Some of the other industrial processes in which bacteria play a part are: the fermentation of indigo by the ferment, indican, produced by *Bacillus indigogenus;* the use of cultures of lactic-acid bacteria in distillery mash; the commercial production of lactic acid; the manufacture of certain white and Belgian beers; and in association with yeasts in the preparation of ginger beer.

Dairy Manufacturing.—Although bacteria are responsible for many faults in dairy manufacturing, they perform valuable services in the preparation of milk products. Artificial buttermilk is made by inoculating milk with a previously soured milk or with a pure culture of lactic-acid bacteria. Manufacturing pharmacists have prepared tablets consisting of mixtures of milk sugar and dried cultures of lactic-acid bacteria for use in making Bulgarian soured milk. Dutch or cottage cheese may be prepared from milk that has spontaneously soured or has been soured by the addition of cultures.

Butter may be made from cream which has ripened spontaneously but, when so made, is likely to vary in quality and uniformity. Uniform results are obtained by the use of starters which are cultures of the

common lactic-acid bacteria and some other closely related organisms, either natural starters or pure-culture starters.   The use of the starters tends to suppress the growth of undesirable bacteria and produces a product of desirable flavor and aroma.

Many different kinds of cheese are prepared from milk by the coagulation or curdling of the protein followed by the more or less complete removal of the whey.· Bacteria and in some cases fungi are active in bringing about the changes in the ripening process.   Cheeses may be classed as acid curd and rennet curd, the latter including hard and soft types.   The first includes the Dutch cheese used without ripening and already noted and a modified type which is ripened.   English and American Cheddar, Swiss, and Dutch Edam are types of hard cheeses, and Limburger and others are types of the soft cheeses.   Both chemical enzymes and bacteria produce changes during the ripening processes, but the latter are the important agents in the production of flavor as a result of their decomposition products.   Soft cheeses in relation to microorganisms may be classed as follows: (1) those in which the ripening is caused by bacteria alone as in Limburger, Backstein and some others; (2) others in which molds contribute to the ripening as in Brie, Camembert and others; and (3) still others in which the molds play the most important part as Roquefort, Gorgonzola and Stilton.

The Preparation of Plant Products.—Tobacco leaves after sweating and after they become "shed ripe" are made to undergo fermentation. This fermentation is due in large part to the action of bacteria.· The use of pure cultures of bacteria for producing desirable changes in tobacco has been reported, giving especially an improvement in flavor and aroma. *Ensilage* or silage is prepared from chopped or shredded green forage, either a single crop like corn or sorghum or mixed with other succulent vegetable materials, or the others may be used without corn.   The chopped material is packed into a tight structure, or silo.   Since materials are fresh and green when packed, the first changes are caused by the activity of the living protoplasm and enzymes, but the most of the fermentative changes are produced by bacteria of the lactic-acid group which suppress or inhibit the growth of other organisms.   In some cases uniformity of product has been secured by inoculation with cultures of lactic-acid bacteria.   In the preparation of *brown hay* the grasses or clovers used are made into a compact stack, well trodden down and thatched to shed rain.   Soon spontaneous heating takes place for some days and after 6 to 10 weeks the finished product is a firm dry mass of pale or dark brown color.   Bacteria bring about lactic and butyric fermentations and other changes which give a pleasant aromatic odor. *Sauerkraut* is prepared by packing shredded cabbage and salt into large jars, casks or barrels.   The cell sap from the cabbage and the salt form

a weak brine which fills all spaces and covers the mass, which is generally held down by a weighted cover. Lactic-acid fermentation is brought about by the bacterial organisms introduced with the cabbage, and many other substances are developed which contribute to the flavor. Other vegetables, such as sweet corn, beets, etc., may sometimes be fermented the same as sauerkraut. Dill pickles also owe their distinctive character to the production of lactic acid which prevents the growth of putrefactive bacteria. A vegetable cheese known as "natto" is made in Japan by fermenting boiled soybeans. At least two different rod forms are concerned in the process, one responsible for the taste and aroma, the other for the mucilaginous, viscous consistency.

The Preparation of Animal Products.—Bacteria are also concerned in the preparation of pickled fish or *sauer* herring, and the lutfisk of the Scandinavian countries. The ripening of game which is caused by the initial stages of putrefaction, gives a flavor which is relished by some people. Bacteria also play an important part in the tanning of hides and leather preparation.

HARMFUL RELATIONS OF BACTERIA

Relation to Milk and Dairy Products.—Milk may be contaminated by bacteria from the cow's udder and by others from the soil, air, water, manure, bedding, milk pails and the milker. Most of the species introduced into freshly drawn milk are crowded into the background or disappear entirely by the development of the forms that are normally present: (1) the *lactic ferments*, which change the milk sugar into lactic acid resulting in the spontaneous souring; (2) forms which secrete a rennetlike enzyme capable of curdling the milk, and others capable of digesting the coagulated casein so as to leave a clear transparent liquid; and (3) forms which cause neither souring, curdling nor the digestion of the casein and which may be present in considerable numbers without affecting taste or appearance.

When certain other forms of introduced bacteria gain the upper hand, milk may be made unattractive in appearance, have an objectionable flavor or odor or even be dangerous to the health if used for food. Some of these more common milk faults or diseases are: (1) *Blue milk*. This is characterized by bluish spots on the surface which later become confluent and is caused by rod-shaped, aerobic bacteria, *Bacillus cyanogenus*. The pigment is soluble and diffusible and so may in time diffuse through the liquid. (2) *Red milk*. Several microorganisms may cause a red coloration, but their action is slow and only becomes evident on milk that is several days old. The most important species are *B. prodigiosus* and *B. lactis erythrogenes*. Besides the forms causing blue or red milk various other chromogenic forms have been recorded as responsible for yellow,

orange, amber, green or violet colorations. (3) *Bitter milk.* Bitter flavor in milk may be caused by certain plants consumed by the animals, but many such cases are due to bacteria. In certain cases the bitter flavor is caused by decomposition products of the casein. (4) *Ropy or slimy milk.* Ropy or slimy milk may result from a diseased udder and should not be confused with a somewhat similar effect caused by bacteria. In the bacterial type, the milk in 12 to 30 hours after standing may be drawn out into long, viscous threads. A number of different species of bacteria are responsible for these changes, but especially *B. lactis viscosus.*

It is generally agreed that bacteria play a prominent part in the development of rancidity in butter, but under certain conditions some of the fungous flora may play a part. Lack of flavor may be due to the absence of the proper flavor-producing organisms. Some of the undesirable characteristics or butter faults different from lack of flavor or from rancidity are: (1) *Putrid butter.* This has been shown to be caused by several different species of putrefactive bacteria. (2) *Turnip-flavored butter.* Certain bacteria are able to produce decomposition products in butter giving it the odor and taste of turnips, rutabagas or other root crops. (3) *Other off-flavors.* Several types have been definitely shown to be caused by bacteria: "cowy" butter, by a bacillus that produced the odor characteristic of stables; lardy or tallowy butter caused by a lactic-acid form; oily butter, caused by certain sour-milk forms which produce an odor and taste resembling machine oil; and bitter butter caused by the same forms which cause bitter milk.

Cheese is more subject to abnormal fermentations than butter because of its high nitrogen content. Some of the cheese faults caused by bacteria may be noted: (1) *"gassy" fermentations* which cause small holes, or "pinholey" curds, larger holes known as "Swiss holes" or, if the gas is more abundant, "floaters" may form by the curds rising to the surface of the whey. These conditions are caused by the decomposition of sugar of milk in such a way as to produce carbon dioxide and hydrogen and sometimes alcohol. They are known to be caused by a number of different bacteria, notably by *Bacillus lactis aerogenes.* (2) *Putrid or rotten cheese* in which localized spots or extensive portions undergo a putrefactive decomposition, in which the casein is changed into a soft slimy mass, resembling somewhat the normal condition of Limburger. (3) *Other off types.* These include mottled cheese characterized by a wavy or mottled appearance, cheese with a bitter flavor, one type of which is caused by a specific Micrococcus, rusty spot caused by *B. rudensis* and various other colored spots caused by other pigment producers.

Other Food Faults.—The production of ropy or slimy bread under certain conditions is caused by several different species of bacteria. The interior of affected loaves may become soft and slimy and sufficiently

viscous to be drawn out into long threads when the loaf is broken apart. Many of the cases of spoilage of home-canned vegetables are caused by imperfect sterilization which permits certain sporeformers to develop. The occurrence of "swelled cans" from the factory products is not uncommon, and may result from the development of anaerobic sporeforming species that may not be killed in the canning process. Much of the spoilage of raw or cooked vegetables and meat and meat products is due to bacteria, which may be much retarded in their development by commercial refrigeration and home iceboxes and electric refrigerators. Mention may also be made of the spoiling of eggs, sometimes with putrefaction accompanied by the formation of sulphureted hydrogen (H_2S) and sometimes without the ill-smelling gas. Certain fungi also play a part in the spoilage of eggs. Bacteria are also responsible for ptomaine poisoning, two types of which are recognized: (1) by eating foods which contain various amines as decomposition products of amino acid, some of which are very poisonous; and (2) by eating food which contains true toxins or endotoxins produced by specific bacteria, especially *Bacillus botulinus*, and not by the decomposition of protein as in the first case.

Faults of Beverages.—These occur in both fermented and distilled beverages but are more common in the former. A few of these abnormalities caused by bacteria are: the turning of wine or beer with the formation of a lactic-acid or a vinegar taint, the ropiness of wine from the growth of species which develop a slime; the loss of color in wine, the mannitic fermentation of wine by the decomposition of the sugar into mannite, the bittering of wine, the turbidity of beer caused by certain Sarcina forms and the butyric-acid fermentation of distillery mash by *Granulobacter saccharobutyricum*.

Bacteria as Weeds of the Sugar Refinery.—Gelatinous masses may appear in juices of sugar beets, sugar cane, sorghum or other plants used in the manufacture of cane sugar, and may develop under certain conditions in such quantity as to be very injurious. This condition is caused by the growth of a mucus-forming bacterium, *Streptococcus mesenteroides*, which uses some of the sugar in its own nutrition thus developing the mucus, and changes some into grape sugar, thus increasing the proportion of molasses. Another weed of the sugar refinery is *Clastridium gelatinosum* which decomposes the sugar with the formation of butyric acid and some other substances. Other species derived from the air, water or soil may produce organic acids and sometimes large amounts of gaseous products. Explosions in sugar refineries have been recorded owing to the formation of marsh gas.

Diseases of Man and Animals.—A very high percentage of the infectious diseases of man and animals are caused by specific bacterial organisms. A study of these diseases constitutes the major portion of the

subjects of medical bacteriology and veterinary bacteriology. Prominent among the bacterial diseases of man are superative infections and blood poisoning, pneumonia, spinal meningitis, gonorrhea, tetanus, diphtheria, plague, typhoid fever and related troubles, tuberculosis, cholera, leprosy, syphilis and yellow fever. The more important bacterial diseases of domestic animals are hemorrhagic septicemia in birds, cattle and swine anthrax, blackleg, Johne's disease, bovine tuberculosis, tetanus, glanders or farcy, swine plague, contagious abortion, fowl or chicken cholera and white diarrhea of chicks.

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CHAPTER IV

THE RELATION OF PLANT DISEASES IN GENERAL TO HUMAN AFFAIRS

HOW PLANT DISEASES CAUSE INJURY OR LOSSES

General Effects of Diseases.—Diseases which affect the aerial parts of plants may cause localized or general disturbances, which result in the killing of portions of leaves, or entire leaves, or only in a general reduced functional activity. One of the most important of the life processes to suffer derangement is the manufacture of carbohydrate foods by the process of photosynthesis. This function may be greatly retarded or reduced in production by the loss of active leaf tissue by spotting, by leaf fall or by reduction in general chlorophyll reconstruction which leads to chlorosis or yellowing of the foliage. Localized injuries to twigs and branches may have an indirect effect upon the foliage of distal parts. Both foliage and twig or branch disturbances may cause injury indirectly to flowers or fruit, but many of our serious diseases of such structures are due to direct invasion by parasites. Functional disturbances or parasites operating through the root system may have an indirect effect upon the aerial parts, and similar disturbances affecting the aerial parts may lead to impairment of root functions. The interdependence of parts is such that any deep-seated disturbance of function in aerial parts must affect the subterranean parts and vice versa. Brief consideration may be given to the types of injury and the way in which plant diseases cause loss.

The Killing of Annuals and Perennials.—From the time the seed germinates in the soil and throughout the balance of life the plant may suffer from the effects of either parasitic or nonparasitic disturbances. Plants in their juvenile condition or as seedlings, whether annual or perennial, are especially susceptible to unfavorable influences, many of which have little effect after some stage of maturity has been reached. Certain other diseases are able to attack seedlings and continue their inroads upon older plants and perhaps reach their greatest severity as producing maturity is approaching. In our annual crops such as cereals and other field crops, forage crops, garden vegetables, etc., killing during the juvenile stage may result in thin stands or even such a complete kill as to require reseeding or replanting. In other cases premature death may come at varying times before maturity, but frequently sufficiently

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early to prevent the harvesting of the desired crop or to reduce the yield. This may be illustrated by the killing of nearly mature cotton plants by wilt or by root rot or the killing of tomato plants by curly top. In cases like tomato curly top, the killing may occur before any fruit has been matured; in other cases, the killing may occur only late in the period of production. In the most severe cases, a complete crop failure may result.

While the killing of annual plants is serious for many crops, the loss of perennial plants like orchard crops, or forest trees which require a long period to reach the producing stage is even more serious. It is possible for winter injury to destroy established orchard trees in a single season. In one western orchard area after a severe winter, numerous twenty-year-old orchards were nearly wiped out of existence. In contrast to such losses the loss of an annual crop sinks into insignificance. A fungous disease such as mushroom root rot may not work so rapidly, but cases are on record in which nearly one-fourth of the trees of an established orchard have been killed in four years. In certain severe epiphytotics of fire blight before the present control practices were adopted, entire pear orchards were wiped out and their culture never resumed in those areas, notably the Gulf Coast of Texas. A disease such as peach yellows, which is prevalent in the eastern United States, may cause very heavy losses. Cases have been recorded of a 50 per cent kill in three to four years and of even a loss of 94 per cent of the trees in a period of six years. It is stated that losses of 1 to 3 per cent annually are common for even the quiescent periods between epiphytotics. The completeness of the destruction of chestnut forests of eastern United States by the chestnut-tree blight is without parallel in the annals of plant pathology. It has been making a clean sweep, killing young growth, merchantable timber of the forests and the beautiful shade trees of cities and country estates. It is difficult for one who has not witnessed the ravages of this disease to appreciate the thoroughness of the destruction. Even such a perennial crop as alfalfa may be so thinned by the ravages of a single parasite or by the combined action of several parasitic and nonparasitic agencies that its retention is no longer profitable.

The Reduction in Productiveness of Perennials.—Certain diseases of perennial plants may not be sufficiently virulent to kill the affected plants outright but, rather, leave them to grow in a dwarfed or crippled condition for a period of years, sometimes completely ruined from the standpoint of production or, in other cases, with a greatly lowered yield, perhaps of inferior quality. A planting of raspberries or blackberries may be completely ruined although not thoroughly killed by a general infection with the orange rust. The brambles are not killed by the several virous diseases by which they may be affected but their producing power is greatly lowered. Plum and cherry trees may develop such

numerous infections of black knot that the trees are practically worthless and in certain cases this disease has led to the complete abandonment of the culture of these crops. Similar disastrous effects have resulted from the uncontrolled spread of the plum-pocket disease of plum varieties. Perennial infections in perennials are especially insidious, since the affected plants cannot recover, and the parasite either gains headway or holds its own. The blister rust of the white and other five-needle pines offers a striking illustration of another serious disease of forest trees. The disease may be fatal in a few years to seedlings and young trees, but the progress is relatively slow in mature trees, which may live on through a period of years, becoming crippled, retarded in growth and dying when the main trunk is finally girdled by inroads of the parasite.

Reduction in Yield.—The effect of disease upon the yield of the marketable product may vary all the way from only a slight reduction to a complete failure without causing the death of the affected plants. Many cases of reduced yield as a result of disease pass unnoticed by the grower or the amount of damage is often underestimated. In all crop groups including fruits, truck crops, cereals, forage crops, ornamentals and forest trees numerous illustrations might be cited of greatly reduced yields of the market product from the effect of diseases. A selection of only a few typical cases will be presented.

Apple scab may cause such severe infections on blossoms, fruit pedicels and young fruit as to cause a complete blighting or dropping of the fruit while still very young. This type of injury is frequently overlooked by orchardists who attribute such failures to adverse climatic conditions such as frosts or cold rains at blossoming time. In other cases the percentage of kill is low and may even be of value in reducing the labor of thinning, and, in many such cases, the amount of reduction in set is overlooked. Similar complete failure of fruit to set may result in plums, prunes and cherries from the blossom-blight phase of the brown-rot fungus or in apples and pears from the blossom-blight phase of fire blight. Leaf curl of peach, by direct attacks upon the flowers and young fruit and by indirect effects of foliage invasions, may prevent the setting of fruit or cause it to drop before reaching maturity. In small fruits, such as currants or gooseberries, infections on the fruit or fruit pedicels are responsible for extensive dropping of berries previous to reaching maturity.

The reduction in total yield of root crops may result from either systemic troubles or from localized parasites, either on aerial parts or on the roots or other subterranean parts. The reduction in yield by a systemic disease may be illustrated by the effects of the virous disease of the potato known as leaf roll. Reductions in yield from this disease have been reported from foreign countries and various portions of the United States and Canada to vary from a minimum of 14 per cent to a maximum

of 97.6 per cent. Rugose mosaic of the potato, another virous disease, has caused the yield in five years to drop from 226 to 167 bushels per acre at the beginning of the tests to 29 to 13 bushels per acre or even to complete failures. Even in a disease such as potato scab which is localized largely upon the tubers, the use of untreated scabby seed or growing the crop on contaminated soil may cause a reduction equal to from one-fifth to one-sixth of the crop as contrasted with the yield when free from scab.

Cereals may suffer serious reductions in yields from smuts and rusts as well as marked reductions of grade or quality which will be referred to later. The amount of bunt in wheat may vary from traces only with no appreciable effect on yield to farm seedings showing 80 or more per cent of the heads smutted in some cases when seed disinfection is not practiced, and 20 to 30 per cent smut is not uncommon under certain conditions even with carefully treated seed. The reduced yield in smuts results from the direct invasion of the grains by the smut fungus, while in the rusts, which are foliage parasites, the reduced yields are caused by the indirect effect of the rust fungi upon the foliage. In severe epiphytotics of stem rust of wheat, the grain may be so badly shriveled as to reduce the yield to as low as four bushels of poor quality grain per acre. In the corn belt of the Mississippi Valley, the root, stalk and ear rots of corn are responsible for enormous losses. It has been estimated that the losses caused by these rots, which result from poor stands, stunting of growth and reduced size of ears, would be fully 10 per cent and perhaps more in the corn-belt states. Even nonparasitic factors may result in a complete failure of a cereal crop to mature the grain, although the plants may live through the season. This is illustrated by the failure of wheat heads to fill as a result of frosts at the time when the heads are emerging from the boot. In such cases the wheat may produce no mature grain and makes only a very poor hay crop. A complete failure of winter wheat to head has resulted from early July seedings, owing to the combined effect of high temperatures and long periods of daylight during the juvenile stages.

In our forests various diseases, wood-destroying fungi or other agencies slow down the rate of growth and, consequently, reduce the laying down of wood, or in other cases cause a destruction or deterioration of a certain percentage of the merchantable volume. These agencies may then reduce the yield of the forest as measured in footage or board feet of lumber. It has been estimated that the total cull from decay in merchantable stands of commercial species of softwood and hardwood trees represent an average of 14.5 per cent.

Lowering of Grade or Quality.—The marketable product from various crops may be lowered in grade or quality from various defects caused by

parasitic organisms acting directly or indirectly or by injuries resulting from nonparasitic agencies. In some market produce, especially fruits and vegetables, a certain percentage of the harvested crop goes into discard as culls because of various defects. This may be illustrated by the defects in apples which are taken into consideration in sorting and grading: (1) *insect injuries*, including worms, unhealed stings, healed stings, aphis injury and any other insect-related defects; (2) fungous *lesions*, including scab, fruit spots, blotch, sooty blotch, rust, mildew russet and numerous types of rot or decay; and (3) nonparasitic defects such as bitter pit, drought spot, sunscald, frost injury, spray burn, hail injury, wind falls, limb rub, bruises, cracks, stem punctures, box cuts, undercolor, undersize, etc. Some of the specified defects throw the fruit into the culls at once while for others a certain tolerance is established for different commercial grades. Some of the prestorage defects may continue to develop after storage or become more pronounced during the storage, while entirely new troubles may make their appearance. In the other fruit crops also certain percentages of the harvested crop are discarded as culls, the detailed defects varying with the crop.

The defects in potatoes which should be taken into consideration in sorting and grading of the harvested tubers, some of which become more in evidence during storage are: (1) *insect and nematode injuries* including the work of the tuber moth, wireworms, flea beetles, eelworms, and any other forms of animal life; (2) *fungous or bacterial lesions*, including various dry and soft rots, surface defects caused by common scab, powdery scab, wart, black scurf or Rhizoctonia and silver scurf, and internal necroses by wilt fungi (Fusarium and Verticillium); (3) *virous disease injuries*, including spindle tubers and other off shapes, and some internal necroses (net necrosis); and (4) *nonparasitic defects* such as chemical (salt) injuries, drought and heat necroses, enlarged lenticels, freezing injury, greening or sunburn, growth cracks, knobbiness and second growth, hollow heart, immaturity, internal brown spot, scald and mechanical injuries. As in fruit crops some of the defects throw the tuber into culls while for others a certain tolerance is permitted in the market grades. In the other truck crops diseases may be responsible for many discards at market time and for the lowering of grade or quality of the packed product.

The value of grasses, cereal crops or legumes for hay may be greatly lessened by the attacks of foliage diseases, which either kill extensive areas of leaf tissue or cause much shattering during cutting and handling. This injury is well illustrated by the leaf spot and the yellow leaf blotch of alfalfa (*Pseudopeziza medicaginis* and *Pyrenopeziza medicaginis*). In these troubles in severe attacks, many of the lower leaves will have fallen to the ground before the time of cutting, while many others which

were less affected will shatter off during the curing and handling of the hay. Sometimes the final product from such fields is little more than a mass of naked stems, while the really valuable portion, the leaflets, has been left behind in the fields. The value of such crops as hay is lessened not only by the losses from shattering, but the nutritive value of the remaining portions is appreciably lowered.

The lowering of grade or quality of the products from forest trees is produced by the work of wood-destroying fungi, especially heart rots. The value of timber for the various purposes whether for lumber, posts, poles or paper pulp is lessened by incipient decay, and, consequently, the various forest products may suffer a reduction in commercial grade, while timber showing too serious injuries may be left in the forest as a total loss.

The Destruction of the Market Product Previous to Harvest.—Various diseases may be localized in the merchantable portions of our various crop plants and destroy the market article by decay or replacement, either by a slow development during the maturing period or by sudden onset and rapid advance in the period of late maturity. Fruit crops, truck crops and other crops represented by fruits, tubers, bulbs, corms, fleshy roots or other storage organs which are reserves of food material are especially subject to rapid decay. Some of these preharvest rots may continue in the harvested crop and cause heavy losses during storage or during transit to market.

Two outstanding illustrations of destructive fruit diseases may be noted. Bitter rot under favorable seasonal conditions is the most ruinous of all apple diseases in the regions of its maximum severity, and, before the days of effective control it was responsible for enormous losses. Some idea of the destructiveness of the disease may be gained from statements made by workers in the bitter rot areas at various periods: "the promise of spring ends in disappointment and decay" (1870); "this season a destruction of 50 to 75 per cent of the crop" (1889); "orchards which in midsummer promised a yield of 25,000 barrels of choice apples, produced only about 5000" (Missouri); in favorable seasons "orchards in which sources of infection are present may lose the entire crop because of bitter rot" (1918). The promise of a bountiful harvest may be blasted in a few days by the sudden onset and rapid advance of the disease. The brown rot of stone fruits, peaches, plums, prunes and cherries, may cause heavy losses in the orchard previous to harvest and this disease becomes of much importance after the crop leaves the orchard. In some peach sections of the south losses as high as 50 per cent of the crop have been reported, in seasons of epiphytotic severity.

The smut diseases of cereals, especially of wheat, oats and barley offer striking illustrations of the destruction of the market product, the

grain, previous to harvest. This feature for bunt or stinking smut of wheat has already been emphasized under reduction of yield.

Losses in Storage or in Transit to Market.—Certain diseases of fruits and vegetables begin their attacks in the field previous to harvest and continue to develop during storage or during transportation to market, while certain others are confined entirely or almost exclusively to the harvested crop and make their appearance at various stages of the storage period. These losses are of concern to farmers, the packers, the commission men, the railway companies, retailers and the consumers. The losses can only be reduced by the combined efforts of all agencies concerned: by attention to control practices for the maturing crop, care in harvesting, providing sanitary conditions for sorting and packing, control of temperature conditions during storage and transport to market, and proper handling after reaching the market. It is beyond the limits of this discussion to mention the numerous parasitic and nonparasitic diseases which are of importance during storage or in transit to market. Storage and transportation losses may be heavy (*Penicillium spp.*, etc.) in citrus and in other subtropical fruits such as pineapples and bananas; in apples and pears, especially from blue mold (*Penicillium spp.*) and gray mold (*Botrytis cinerea*); in stone fruits by brown rot (*Sclerotinia spp.*); in strawberries by leak (Rhizopus and Pythium) and by gray mold (*Botrytis cinerea*); in vegetables such as asparagus, peppers, carrots, beans, cabbage, lettuce, etc. by bacterial soft rots (*Bacillus carotovorus*), Sclerotinia rot (*Sclerotinia sclerotiorum*) and leak (Pythium and Rhizopus); in beans by anthracnose (*Colletotrichum lindemuthianum*); in cabbage and other crucifiers by black rot (*Bacterium campestre*); in potatoes by blight (*Phytophthora infestans*), Fusarium rots (various Fusaria); in sweet potatoes by soft rot (*Rhizopus nigricans*); and in tomato by anthracnose (*Colletotrichum phomoides*) and Phoma rot (*Phoma destructiva*). The above list represents some of the storage and transportation troubles of outstanding importance.

The losses in storage or in transit to market or while being held at terminals may result from: (1) the loss of the product, varying from slight to complete; (2) from the bill for transportation or storage which will fall as a loss to either the shipper, agent or carrier depending upon the establishment of responsibility; and (3) in the expenditure of time and money in sorting, reconditioning or repacking if the decay is not complete before the produce can be offered for sale. The ordinary consumer has but a faint realization of the magnitude of these losses. Many illustrations might be cited but space will permit only a single specific illustration. Records on shipments of apples from Washington to eastern terminals for the period from 1930 to 1935 showed heavy losses from blue mold, the highest average percentage of blue mold in carlots being 25, 55, 60, 25

and 40, with the highest percentage of decay in box lots amounting to 100, 90, 90, 80 and 80. Transportational losses from various rot-producing fungi may reach even higher figures in the shipment of soft fruits and the succulent or fleshy vegetable crops, some of the heaviest losses occurring in shipment from southern regions to northern markets.

Effect of Plant Diseases upon Man and Animals.—Since prices of our agricultural products are influenced by supply and demand, prices of farm commodities may be affected by the prevalence and severity of diseases. The effect of epiphytotics of stem rust of wheat may be reflected in the higher market quotations. The sharp advance in the price of citrus fruit has followed the recent serious losses to this crop from one of the most disastrous freezes in California history. In past time certain plant diseases have had more disastrous and far-reaching effects than in current times with our better knowledge of methods of control. One of the most notable cases is the epiphytotic of the late blight (*Phyto-phthora infestans*) of the potato which reached its culmination in 1845. The disease was very severe in the northeastern United States and Canada and in continental Europe, but its ravages were more general and complete in Ireland. "The staple food supply of an entire people was destroyed by disease with a suddenness and a completeness unparalleled in the recorded history of mankind. All day the hungry peasant dug his fields to find enough tubers among the soft and stinking masses to make his simple evening meal. The suffering was intense. In spite of the most generous governmental and private aid a quarter million Irish people died of hunger or of the fever resulting from a lack of food" (Whetzel, 1926–1927).

It is unusual for a plant disease to assume such importance as is true for the ergot of rye, which is a destructive disease of this and other cereals, furnishes a drug of world-wide importance, and even at present is responsible for convulsive and gangrenous ergotism in man and animals. The history of European countries is replete with numerous records of fatal results, especially among the poorer classes, from eating bread prepared from the flour of ergotized grain (see Ergot, Chap. VIII).

Some Effects of Diseases upon Land and Property Value.—In addition to diminishing the yields and quality of the products of our fields and forests, the prevalence of certain diseases may cause a depreciation of land value. This is likely to be true if the affected crop is a high-money crop or is one which is better suited to the locality than any other. These plant-disease fungi which affect land value are in most cases soil-borne or soil-inhabiting fungi which may remain in the soil for a long period of years and in some cases can never be eradicated. Many cases might be recorded of the depreciation in land value to the extent of 50 per cent or more from the invasion and establishment of a plant disease which

is either ineradicable or entails the expenditure of enormous sums of money for its suppression or extermination. This kind of injury may cause still heavier losses if the crop to be discarded is one which requires a heavy outlay of money to prepare for its culture. The invasion of the extensive white pine forests of the Pacific Northwest by the blister rust can not fail to cause a depreciation of the value of these forest lands. Lands within a smelter zone are lowered in value from the injury to growing crops from sulphur dioxide which is poured out over the surrounding country. Decrease in land value has resulted from long and persistent drought conditions and frequent dust storms as may be illustrated by the present situation in portions of the northern Mississippi Valley. In certain cases the prevalence of a disease may cause the dismantling of expensive factories as may be illustrated by the failures of sugar-beet culture in eastern and central Washington because of the extreme severity of the curly top disease.

The Cost of Disease Prevention or Control.—Plant diseases require the expenditure of much human effort and the outlay of enormous sums for their control, eradication or exclusion. To the plant-disease account must be charged the cost of machinery or appliances used in seed disinfection, spraying and dusting, soil disinfection, orchard heating and many of the special protective or cleaning treatments used in preparing fruits and other products for the market; also an enormous bill for therapeutants for use in seed and soil disinfection and the protective spraying of growing crops.

The cost of the therapeutants for treating the world supply of seed wheat for bunt or stinking smut of wheat may be used as an illustration. The annual yield of wheat for the world is estimated at 4,000,000,000 bushels. Assuming that the average yield per acre is 15 bushels, this would require 266,000,000 acres for its production. If only one bushel of seed were used per acre and all of this seed were treated with a standard dust disinfectant at $2\frac{1}{2}$ cents per bushel, the cost of the disinfectant alone would amount to over six million dollars. If similar figures could be computed for the crops which require much more expensive prophylactic measures, they might throw some light on one of the important causes of farm mortgage foreclosures.

EXTENT OF PLANT-DISEASE LOSSES

There is a general agreement of producers and scientific investigators that plant diseases and insect pests are responsible for enormous losses each year. Attempts have been made in various countries and at various times to put these losses into definite figures using two different methods of presentation: (1) the actual loss in dollars to the producer from decreased yields or market losses; and (2) the quantity or the per-

centagè of the total crop lost or destroyed. It should be realized that in both of these methods the figures at best are only an approximation of the true losses, perhaps underestimated in some cases and overestimated in others.

Some of the various figures representing the money losses may be presented first:

Crops	Disease	Country and year	Amount of loss
Cereals......................	Rusts	Prussia, 1891	$ 45,000,000
Cereals......................	Rusts	Canada, annual	40,000,000
Wheat.......................	Yellow rust	Germany, 1926	75,000,000
Wheat.......................	Stem rust	U. S., 1916	283,000,000
Wheat.......................	Stem rust	Canada, annual	30,000,000
Wheat.......................	Stem rust	U. S., average 1915–1924	55,000,000
Wheat.......................	Bunt	U. S., annual	11,000,000
Wheat.......................	Loose smut	U. S., annual	3,000,000
Apple.......................	Scab	N. York, annual	3,000,000
Apple.......................	Bitter rot	U. S., 1900	10,000,000
Peach.......................	Leaf curl	U. S., 1900	2,335,000
Peach.......................	Brown rot	U. S., annual	3–4,000,000
Bean.......................	Anthracnose	Mich., 1917	3,000,000
Chestnut....................	Blight	U. S., 1906–1911	25,000,000

It may be noted that some of these figures represent losses in years of extreme epiphytotic severity, while others are estimates of annual losses. The figures given for the chestnut-tree blight represent only the first few years of the spread of the disease which has since continued unchecked.

In the table on page 51 the reduction in production from the inroads of all the diseases affecting the specified crops in the United States is presented in comparison with the total production as recorded in the Plant Disease Survey Reporter for the years 1934 and 1939. In most of the crops listed, total production has increased in 1939 with a parallel reduction in yield from disease. In the case of corn and sweet potato, however, with greatly increased yields, a marked reduction in percentage of loss was noted in 1939, but with the peach, diseases appeared to be exacting a higher toll than in 1934.

Rusts and smuts cause about one-half of the wheat losses, the two smuts take the heaviest toll in barley, rusts and smuts are the most destructive for oats, and the ear and stalk rots are the most destructive of the corn diseases. Storage rots of the sweet potato cause more loss than all the field diseases, mosaic and leaf roll are responsible for the heaviest losses in Irish potatoes, Fusarium wilt and blossom-end rot

take the heaviest toll in market tomatoes, bacterial blight is the most serious for snap beans, while root rots are the most serious diseases of peas. Scab is the disease of outstanding importance for apples, fire blight takes the heaviest toll of pears, while brown rot and scab are the most serious for peaches.

Crop	1934		1939	
	Total production, bushels	Total reduction, bushels	Total production, bushels	Total reduction, bushels
Wheat..............	496,469,000	21,125,000	754,971,000	44,376,000
Barley..............	118,929,000	3,535,000	276,298,000	10,565,000
Rye................	16,040,000	278,000	39,249,000	333,000
Oats...............	528,815,000	47,090,000	937,215,000	104,556,000
Corn...............	1,380,718,000	213,570,000	2,619,137,000	198,913,000
Sweet potato........	67,400,000	8,385,000	72,679,000	3,417,000
Irish potato.........	385,288,000	70,948,000	360,992,000	59,124,000
Tomato (market).....	18,080,000	3,063,000	24,585,000	2,120,000
Snap beans (market).	13,486,000	1,645,000	22,627,000	Data incomplete
Green peas (market)..	7,442,000	172,000	9,627,000	Data incomplete
Apple..............	119,855,000	8,112,000	100,284,000	7,241,000
Pear...............	23,474,000	565,000	30,910,000	726,000
Peach..............	45,404,000	1,682,000	61,730,000	2,469,000

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CHAPTER V

THE DISSEMINATION OF PLANT DISEASES

The spread of parasitic or of virous diseases from plant to plant or from one locality to another involves either the transport of the pathogene or of its spores or of the viruliferous principle. A knowledge of the methods of dissemination is of fundamental value in determining and understanding methods of control.

Air and Wind Dissemination.—The spores of many different species of fungi are prevalent in the air and some of these represent parasitic species. Two general types of fungous spores are adapted to air and wind dissemination: (1) those borne singly or in chains on the ends of aerial conidiophores from which they are easily detached, or others that are liberated from the fruiting body as a dry, powdery mass; (2) those which are separated from the spore fruit by some explosive mechanism which forces them into the air, after which they may be carried away by air currents. This prevalence of spores in the air has been demonstrated by exposing plates of culture media to the air and observing the fungi which develop; by filtering air through a trap which retains the spores so that they may be observed under the microscope or identified by planting out in culture media; by the use of spore traps which collect the spores which are set free by an explosive mechanism and carried away by the wind or by convection currents; and by direct microscopic examination or by the use of a light beam. In addition to the *direct* dispersal of spores by air currents, the wind may assist in the *indirect* spread of plant diseases by transporting diseased parts of the host, especially infected leaves or flower parts blown from the diseased tree or later picked up by the wind from the ground litter beneath diseased trees. It may be noted also that wind-blown rain may sometimes transport pathogenic bacteria or fungi which have no special devices for air or wind transport.

Typical examples of fungi adapted to air or wind dissemination of the spores without the use of an explosive mechanism are to be found in many forms of Hyphomycetes such as Penicillium, Aspergillus, Alternaria, Verticillium, etc.; in the conidial stages of the white rusts, downy mildews and powdery mildews; in certain spore stages of smuts and rusts; and in the imperfect stages of some sac fungi or ascomycetes.

Two distinct types of forcible ejection of spores are illustrated: (1) By ascus fruits from which the ascospores are forcibly expelled by the explosion of the asci. Two different types of ascospore expulsion are to be found: (*a*) the successive explosion of the spore sacs from a perithecium until the supply is exhausted, either the entire eight spores shot out at once or in succession like a repeating rifle; and (*b*) the simultaneous explosion of a large number of asci, characteristic of apothecia, giving rise to the phenomenon of "puffing" with the production of visible clouds of spores. Numerous forms of fungi from the Pyrenomycetes including both parasitic and saprophytic species show the first type of forcible expulsion of their ascospores, for example, the powdery mildews, the apple-scab fungus, and the chestnut-tree blight pathogen; while the second type is characteristic of both parasitic and saprophytic species of Discomycetes, for example, the Sclerotinia of wilt, the Sclerotinia of brown rot of stone fruits, the Pseudopeziza of clover and alfalfa leaf spot, the Rhytisma of tar spot of maple, the Coccomyces of cherry leaf spot and others.

(2) By the basidium fruits which provide for the forcible separation of the basidiospores from the basidia. In this case the four spores borne on each basidium are ejected in succession, each spore being thrown off by a jerking process due to the release of hydrostatic pressure. This type of spore discharge is characteristic for the sporidia of the smut fungi, for the sporidia produced from the promycelium of rust fungi, well illustrated by the cedar-rust fungus and other species; and by the various species of fungi producing definite basidia-bearing sporophores or fruiting bodies (see Fig. 26).

Dissemination by Water.—Water may serve for the dissemination of pathogens in two ways: (1) as a medium in which actively motile organisms or spores may swim about; and (2) by the mechanical action of runoff of rain, the flowing of irrigation water, or by stream flow.

Actively motile cells are produced by some bacterial pathogens, the water molds, the chytrids, the white rusts, and some of the downy mildews. Such forms depend wholly or in part upon liquid moisture as a medium in which they may develop and through which they may migrate by the action of their cilia or flagella. Liquid moisture is required for the stomatal or water-pore infection by certain bacteria (see Black Rot of Cabbage). The chytrids are primarily aquatic-forms but even forms affecting land plants must rely on liquid soil moisture for the dispersal of their swarm spores, for example in the crown wart of alfalfa, potato wart and clubroot of cabbage and other crucifers. This explains in part why damping-off by *Pythium debaryanum* is favored by a wet soil. Among the white rusts and downy mildews which develop only on aerial parts, the aquatic habit is still retained in part and liquid moisture is

necessary for the formation and dissemination of their swarm spores. This is well illustrated by the late blight of the potato, the white rust of crucifers and the downy mildew of grape.

The dripping of moisture from heavy dews or the runoff from rains may transport nonmotile spores: (1) those which have been lodged on plant parts by wind or other agencies; and (2) those which are residual in origin but are held together by a mucilaginous matrix in which they are embedded. This second condition is illustrated by bacterial exudates as in fire blight of pome fruits and in the olive knot; and by the spore horns or tendrils, extruded from many pycnids or by the gelatinous spore masses formed from acervuli. This production of spores in gelatinous masses is well illustrated by the conidial stage of the chestnut-tree blight fungus and the acervular stage of bean anthracnose. In all of these cases the gelatinous matrix in which the bacteria or the spores are embedded, is dissolved by rains and the separated spores washed down by mechanical action.

Flood water, irrigation water or stream flow may be responsible for the spread of pathogenic forms. Overflow of streams has spread the root and crown rot of celery, while the cultural practice of flooding rice or cranberry fields spreads the disease-producing organisms. Even in row irrigation, the water may carry a load of spores and so increase the chances of infection as has been demonstrated for the leaf spot of sugar beets and the late blight of celery. Dissemination by stream flow is well illustrated by the forms of ergot affecting aquatic grasses in which the sclerotia are "floaters" and so are carried down stream to new hosts.

Dissemination by Insects.—Insects may act simply as carriers of infective material adhering to their bodies, or they may harbor the inoculum within their bodies, and in certain cases make the inoculation by feeding wounds, either by chewing or sucking mouth parts. Both bacterial and fungous diseases are disseminated by insects but they are of outstanding importance as the vectors of virous diseases. In relation to insect dissemination the following groups of plant diseases may be recognized:

1. Organism carried externally and infection accidental, without direct inoculation by the insect. Examples: fire blight of pear and other hosts and bacteriosis of walnut; rusts and smuts of numerous hosts, bitter rot of apple, chestnut-tree blight, late blight of potato, Cercospora leaf spot of beet, cotton anthracnose and many others.

2. Organism or a virus carried externally and infection by mechanical transfer through feeding by the insect carrier. Examples: fire blight of pear and apple, bacterial wilt of Solanaceous plants, black rot of crucifers and some other bacterial diseases; downy mildew of Lima bean, brown

rot of stone fruits, bud rot of carnations, blackleg of cabbage and many other fungous diseases; and certain virous diseases carried by biting insects.

3. Organism not carried by insects, but infection through wounds made by insects. Examples: bacterial leaf spot of cotton and of sugar beet; onion blight; powdery mildew of grasses; chestnut-tree blight and others.

4. Organism carried internally by insects, but without any specific biological relation, that is, the carriage is purely mechanical. In many cases these same organisms are also carried externally. In this mechanical internal transmission the spores remain viable after passage through the alimentary tract but do not increase in number. This behavior is illustrated by the spores of the Sphacelia stage of ergot which have been shown to survive passage through the digestive canal of small arthropods and gastropods.

5. Organism or the infective principle carried internally in the body of the insect carrier and undergoing a multiplication or increase, sometimes only after a definite incubation period. This method of transmission has been proved for certain bacterial diseases but is of outstanding importance for many virous diseases. Among the bacterial diseases falling in this class are the bacterial wilt (*Bacillus tracheiphilus*) of cucurbits carried by the striped and 12-spotted cucumber beetles, and the olive knot (*Pseudomonas savastanoi*) carried by the olive fly. The majority of virous diseases, especially those transmitted by sucking insects belong in this group. Further details of this relation will be found in the chapter on virous diseases.

Dissemination by Other Animal Life.—The other forms of animal life credited with the dissemination of diseases are nematodes, slugs or snails, birds and wild or domestic mammals. Specific cases are the spread of bacterial brown rot of potatoes by nematodes, black rot of cabbage and other crucifers by slugs, the chestnut-tree blight by sapsuckers which drill into the bark, certain wood-rotting fungi by mice and squirrels, and potato scab and wart by manure from horses, cows, or pigs fed on the diseased tubers. It has also been pointed out that the spores of certain parasitic forms mingled with the soil may be carried from contaminated fields to disease-free fields on the feet of domestic animals. Such transport has been reported for potato wart, onion mildew, and soil-borne pathogens of cabbage.

Dissemination by Seed.—As considered in this relation, seed will include true seed, fruits functioning as seed, and also vegetative reproductive structures, such as tubers, fleshy roots, bulbs, corms and rhizomes. A pathogen may be carried *on* or *within* these reproductive bodies or, in some cases, as separate bodies mingled with the seed.

When the infective material is carried only on the surface or mingled with the true seed, the contamination usually occurs during the threshing or separation of the seed. The presence of surface-borne spores can be demonstrated by washing the seed in water, followed by a microscopic examination of the sediment, either before or after concentration by centrifuging. Some of the recorded cases of surface-borne pathogens on seed are the black rot (*Pseudomonas campestris*) of cabbage, the wilt (*Fusarium lini*) of flax, bunt (*Tilletia spp.*) of wheat, covered smut (*Ustilago levis*) of oats, covered smut (*Ustilago hordei*) of barley, stalk smut (*Urocystis occulta*) of rye, millet smut (*Ustilago crameri*) and sorghum and broomcorn smut (*Sphacelotheca sorghi*). Two notable cases of inclusions mingled with the seed are afforded by ergots of rye and other cereals and the seeds of clover and alfalfa dodder that are mixed with the seed during threshing operations.

Some of the diseases known to be carried by the organisms within true seeds are the bacterial blight (*Pseudomonas phaseoli*) of beans, the Phytophthora disease (*Phytophthora phaseoli*) of Lima beans, the anthracnose (*Colletotrichum lindemathianum*) of navy and string beans and many other diseases caused by imperfect fungi. Fruits that function as seeds may harbor the parasite within the seed coats or within the embryo. This has been shown to be true for the anthracnose (*Colletotrichum cereale*) and the scab (*Fusarium roseum*) of wheat, the spot blotch (*Helminthosporium sativum*) of barley and the loose smuts (*Ustilago tritici* and *U. nuda*) of wheat and barley. Among the fruits other than the caryopses of cereals functioning as seeds which are known to harbor the pathogene are beet seeds containing *Phoma betae*, celery seed (mericarps) containing the pycnidia of *Septoria petroseleni* and the seed (achenes) of oyster plant containing mycelium and oöspores of white rust (*Albugo tragopogonis*).

Most of the virous diseases are not carried by the true seeds, but seed carriage is general for some legume mosaics and has been demonstrated for the mosaic of lettuce, the mosaic of petunia and the mosaic of the wild cucumber.

Many causal agents of disease, bacteria, fungi, nematodes, or viruses, are carried on the surface or within the vegetative reproductive structures or storage organs such as roots, tubers, bulbs, corms or rhizomes. The sweet potato and other root crops, the Irish potato, bulbs of the onion, and the corms, bulbs and rhizomes of flowering plants are frequently the carriers of numerous diseases.

Dissemination by Propagating Stock.—Cuttings, herbaceous seedlings, scions used for grafting, and nursery stock may act as carriers of both parasitic and virous diseases. The propagating stock may act simply as a carrier of the spores of a pathogen; incipient or established infections of a

pathogen may be present; viruses may be present and still latent; or visible symptoms may be evident. Infections developed within the seed bed may be carried to the field by some of the transplants. This has been shown to be true for several sweet-potato diseases (Fusarium stem rot, dry rot, and foot rot) at least three different cabbage diseases, and especially for the virous diseases of tobacco.

For fruit, forest or shade trees, or shrubs any diseases which affect the roots or woody stems may be introduced with the young trees. Notable examples are crown gall (*Pseudomonas tumefaciens*), plum black knot (*Dibotryon morbosum*) and chestnut tree blight (*Endothia parasitica*), the latter probably first brought to America on nursery stock imported from China. It is believed that citrus canker and the white-pine blister rust were also introduced with imported nursery stock, the former from Japan and the latter from Europe.

Dissemination by Crude or Commercial Plant Products.—These include such products as fruits, vegetables, grain, hay, straw, packing materials and forest products, that are not used for propagative purposes. Examples may be found in such diseases as scab, black rot and bitter rot of apples, the brown rot of stone fruits, the scab, wart and powdery scab of potatoes and cabbage and other vegetable diseases. Transport of a few other diseases may be noted from recorded cases: gooseberry mildew on packing material; cereal diseases in grain, hay or straw; cereal smuts in meal, bran, flour or contaminated bags; and chestnut-tree blight on logs, poles, posts, ties and tan bark.

Dissemination with Soil, Litter, Compost or Manure.—By the transport of these materials parasitic organisms may be introduced into new environments. The parasites introduced may be either organisms that normally inhabit the soil or they may be accidental inclusions. Damping-off fungi may be brought into the greenhouse with contaminated soil, Sclerotinia wilt of lettuce, tomatoes and other vegetables may be introduced by sclerotia mingled with the soil or with plant refuse, or the use of alfalfa soil for inoculating new fields with legume bacteria may introduce soil-inhabiting pathogens. Numerous other cases of soil contamination by soil carriage or litter have been recorded, including cabbage yellows, flax wilt, carnation wilt, Mycogone disease of mushrooms, black rot, charcoal and stem rot of sweet potato, Rhizoctonia and Fusarium diseases of Irish potato, and the smut of onion.

Forms that are not primarily soil dwellers, may spend a part of their life cycle in the soil or soil refuse. The oöspores of onion mildew (*Peronospora schleideniana*) may be carried with onion leaf litter, cotton anthracnose (*Glomerella gossypii*) on cotton hulls, Ascochyta blight (*Mycosphaerella pinodes*) of peas in pea straw, the leaf spot (*Cercospora beticola*) of beet in the old tops, and, when these are left *in situ* or scattered

over new areas, they may continue the diseases. In none of these cases is there any multiplication of the organism in the soil, but, in the case of corn smut, the spores germinate in the soil or in the compost heap and give rise to numerous secondary spores which may cause a heavy soil contamination.

Dissemination by Other Agricultural Practices.—Plant diseases may be spread by the ordinary operations of transplanting seedlings, by watering, by spraying, by picking or harvesting operations, and by threshing or ginning. A few specific examples may be cited. Blackleg of potatoes may sometimes be spread when cutting seed tubers, but experimental studies have shown this method to be less important than formerly reported; fire blight of pear and apples in pruning; late blight of potatoes during digging and handling; mosaic of tobacco during transplanting or when topping in the field; tomato streak, a virous disease, by tobacco users during the pruning operations; bean anthracnose during cultivation and picking; brown rot of lemons by washing previous to curing; blue mold and other rots of apples by the solutions used for spray-residue removal; covered smuts of cereals by the threshing operation which not only contaminates the sound grain but scatters the smut spores over surrounding fields; and cotton anthracnose by the ginning of the cotton.

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SECTION II

PARASITIC DISEASES

CHAPTER VI

THE CONDITION OF A FUNGUS IN OR ON THE SUBSTRATUM

The Life Phases of Fungi.—The object on or in which a fungus establishes itself may be designated as its *substratum*. This may be the humus of gardens or fields, the dead remains or products of animals or plants, an artificial medium, another living plant or animal or portions of such living organisms—in fact, any material which will furnish the necessary food. Its condition in or on this substratum may be either in one of the *vegetative stages* directed to the procuring (absorbing) of food and the building up of the fungous body, or the purely vegetative structures may have given rise to specialized *reproductive* stages or structures.

Host and Parasite.—If the substratum is a living plant or part of a living plant, it may then be designated as the *host* (also *suscept*) and the fungus as the *parasite* which preys upon it. In its development, the fungus produces more or less serious disturbances in the life of its host; or disease in slight, pronounced or severe form is the result.

VEGETATIVE STAGES OR STRUCTURES

Hyphae and Mycelia.—The typical fungous body consists of a delicate, branched, tubular or filamentous structure, the *mycelium*. These mycelial threads or *hyphae* may be *septate* or *nonseptate* and form an interlacing tangle, or a loose woolly mass or they may be densely interwoven or even compacted into solid bodies. Since certain groups of fungi produce only nonseptate hyphae, while other groups exhibit only septate hyphae, it is, therefore, of importance to determine whether they are septate or continuous. The fungous body of some forms, either primitive or degenerate types, may consist of only a single globular cell no larger than individual cells of a hypha.

A parasitic mycelium may grow on the surface of its host (*external*), or it may be within its host (*internal*). External mycelia generally appear as delicate whitish, cobweblike threads or as sooty brown or black threads making an interlacing tangle on the surface of the host, and they are very characteristic of the powdery mildews and the sooty molds. Internal mycelia, also hyaline or dark, are confined to the intercellular

spaces of the host tissue or penetrate into the interior of the host cells. The presence of hyphae within the invaded host tissues can be demon-

Fig. 15.—Outline diagram of a spore of Penicillium and stages in its germination to form hyphae and a young septate mycelium. (*Adapted from Lafar.*)

strated only by microscopic examinations, unless aggregated into dense masses.

When a mycelium of either parasitic or saprophytic forms begins to spread from a point or focus in which it is established, it shows a tendency to grow radially in all directions, unless interfered with by the character of the substratum. Fungus "fairy rings" are the fruiting bodies developed on or near the periphery of an advancing mycelium, which is concealed within the soil. Many fungous leaf spots are distinctly circular, due to the manner of growth of the internal mycelium, while rotting areas on fruits show a circular surface outline for the same reason. Fungous lesions of the bark of woody hosts are generally slightly elongated or somewhat elliptical, owing to the fact that the lengthwise advance of the mycelium is more rapid than the transverse growth.

Fig. 16.—*A*, young cell of a hypha, showing a highly vacuolate cytoplasm; *B*, an old cell with a single central vacuole and peripheral cytoplasm.

Mycelial Plates or Fans.—In woody hosts and in some herbaceous hosts the mycelium becomes dense and compact and forms whitish pockets or radially or longitudinally elongated white plates or bands which are very evident when the invaded structures are cut open or broken. Such mycelial plates occur within the disintegrating wood of pines and spruces, while in other cases very characteristic tawny sheets

Fig. 17.—The mycelium of the silver-leaf fungus (*Stereum purpureum*) growing on agar.

of mycelium spread out in fanlike forms in the inner layers of the bark or in the cambium (*e.g.*, chestnut-tree blight or mushroom root rot).

Mycelial Strands or Rhizomorphs.—The hyphae of a mycelium are sometimes aggregated to form white, tawny or even dark brown, cordlike or threadlike strands, which vary from slender threads to good-sized strings. The strands generally branch more or less and may frequently fuse to form a network. While these fungous strands may store up some food, they are not primarily storage organs but serve rather to bring the fungus to new hosts or to new parts of the same host and thus widen or

FIG. 18.—Piece of timber infested with the mycelium of *P. sulphureus.* The white masses of fungus fill up the rings and rays produced by their "rotting" action. (*After Hartig.*)

FIG. 19.—Piece of timber completely destroyed by *P. sulphureus*, the mycelium of which fills up the crevices as a white felt. (*After Hartig.*)

enlarge the area occupied.   In this way the fungus may spread from a
single diseased tree to adjacent trees by a natural growth.

Sclerotia or Storage Organs.—Some fungi have developed the habit
of growth of producing dense compacted aggregates of hyphae called

Fig. 20.—Rhizomorphs or fungous strands of the shoestring fungus (*Armillaria mellea*).
(*After Freeman, Minnesota Plant Diseases.*)

"sclerotia" which become filled with food materials in the form of oil
and other compounds.   These structures may be hardly visible to the
unaided eye or as large as a cantaloupe or even larger and are generally
more or less rounded, elongated, cylindrical, globular or ellipsoidal masses,
but sometimes more or less flattened and irregular in form.   In many
cases they are dark colored, either on the surface or throughout the entire

aggregate of cells. In parasitic fungi, they are formed either upon the surface of host parts, concealed between rotting leaves or within internal cavities. (See Rhizoctonia Disease of Potato and Ergot of Rye for examples.)

Sclerotia are, in reality, storage organs filled with the special kinds of reserve food peculiar to the species of fungus by which they are formed. Although they are vegetative structures, they serve essentially the same purpose in the life of many fungi as do spores, for they are able to with-

FIG. 21.—Sclerotia of the wilt fungus (*Sclerotinia trifoliorum*) on alfalfa plants.

stand adverse conditions which would prove fatal to ordinary mycelia. In the sclerotial condition fungi are able to endure extreme desiccation, or long periods of high temperatures or the rigors of winter. Attached to host parts, such as seed or propagating stock, or mingled with the soil or seed, sclerotia are frequently very effective means of the dissemination of parasitic fungi.

REPRODUCTIVE STAGES OR STRUCTURES

The common reproductive structures of fungi are very small bodies of microscopic size, the *spores*, which are either cut off from hyphae or produced by specialized structures known as "spore fruits." Some fungi have retained a very simple method of propagation which does not involve the formation of specialized spores. The production of new cells essentially similar to the parent cell, by a process of budding as in the yeast plant, is a primitive type of reproduction, which is retained by some of our parasitic fungi, notably some smuts, at certain stages in their life cycle.

A. Spores

General Characters.—A spore is generally one to several specialized cells which serve the purpose of disseminating and reproducing the fungus. A single spore under proper conditions of temperature, moisture and host

relations may start the development of a new fungous body, a *mycelium.* Spores function as seeds, serving the same purpose for fungi as the true seeds do for seed plants; they may be either clear or dark, varying from slightly smoky to almost black. The micron or micromillimeter (1/1000 millimeter) is the standard unit for spore measurement, the smallest spore being 1 μ or less in diameter, while in a few cases they may reach 1 millimeter or slightly more (see Fig. 22 for form and septation).

Sexual and Asexual Spores.—Spores may be formed as the result of a breeding act, that is, the union of two separate and distinct cells or

FIG. 22.—Diagrams of various forms of spores.

elements (gametes) which represent male and female. Spores of this type may be classed as "sexual," while those which are formed direct from hyphae without the intervention of a breeding act are "asexual," or without sex.

Kinds of Spores.—The following are the most important spore types: (1) *chlamydospores,* formed by direct transformation of certain cells of a hypha, or of an entire hypha; (2) *swarm spores* or *zoospores,* naked protoplasmic masses of varying form provided with one or more delicate vibratile filaments, the *cilia,* by means of which they are able to swim about; (3) *conidia,* spores of various sizes, forms and septation which are

FIG. 23.—Semidiagrammatic drawings showing types of spores and their manner of formation. *A*, yeast cells, showing primitive method of propagation by budding; *B*, formation of chlamydospores; *C*, chlamydospores formed in a continuous chain; *D*, yeast-like method of production of secondary spores characteristic of some higher fungi, *e.g.*, smuts; *E*, chlamydospores produced at the ends of hyphae; *F*, a sporangium bearing a single nonmotile spore; *G*, a globular sporangium (Mucor type) with numerous non-

pinched off or cut off from the ends of special spore-bearing hyphae, or *conidiophores;* (4) *ascospores,* typically eight in number, (1 to 16) developed within a saclike or club-shaped structure, the *ascus;* (5) *basidiospores* developed on the tips of either two or four (rarely more) slender terminal projections, or *sterigmata,* from a globular, cylindric or club-shaped cell, the *basidium;* (6) *zygospores* formed by the union or *conjugation* of two equal and similar cells or gametes; and (7) *oöspores,* formed by the union (fertilization) of a large, passive, female gamete with a small, active male cell or gamete. The special types of spores peculiar to the rust fungi will be described in the treatment of that group.

B. SPORE FRUITS

General Characters.—In the simpler forms of spore production, the spores are cut off from the ends of specialized, free, aerial branches or conidiophores, or organized within the interior of specialized cells called *sporangia,* these spore-bearing branches being produced direct from the mycelium. With further development, there is the organization of definite complex aggregates of spore-bearing hyphae, frequently surrounded by more or less supporting and protecting tissue. It is these complex aggregates which can with real propriety be designated as *spore fruits* since they are highly specialized structures adapted to spore production, protection and dissemination. Aerial conidiophores and sporangia represent a more primitive condition before the organization of the more complex spore fruits.

Kinds of Spore Fruits.—The following are the most important types of spore fruits: (1) an *aerial conidiophore,* a simple or variously branched, specialized hypha, disposed singly or in groups or tufts, each bearing either a single spore or chains or groups of spores; (2) a *coremium* (pl. coremia) or a dense fascicle of erect conidiophores grouped together to form a sterile stalk and a terminal fertile or spore-bearing head; (3) a *sporangium* (pl. sporangia), or a special multinucleate cell, cut off from the free end of a hypha, and containing an indefinite number of spores, either nonmotile or motile by cilia; (4) a *sorus,* or a small or large mass or cluster of spores borne on short stalks (*e.g.,* sori of white rusts and true rusts); (5) a *pycnium or spermogonium,* a small, flask-shaped body (characteristic of certain rusts) which produces minute, bacterialike bodies, the *pycniospores* or *spermatia;* (6) an *aecium,* a cluster of closely packed, yellow or orange-colored spores, *aeciospores* either naked or in

motile spores; *H,* stages in the formation of uniciliate swarm spores from a sporangium; *I,* a zoosporangium from which biciliate swarm spores have been formed; *J,* types of swarm spores; *K,* stages in the development of a zygospore from the union of equal and similar cells (gametes); *L,* two types of oöspore formation characteristic of white rusts and downy mildews; *M,* four types of origin of conidia; *N,* stages in the development of asci and ascospores; *O, P, Q,* three asci showing different arrangement of the ascospores; *R,* stages in the development of a basidium and basidiospores; *S, T,* two other types of basidia.

Fig. 24.—Semidiagrammatic drawings of types of conidiophores and spore fruits. *A*, a downy mildew, Peronospora; *B*, blue mold or Penicillium; *C*, Aspergillus; *D*, Cercospora; *E*, section of a sorus of white rust (Albugo) with enlarged conidiophores and conidia; *F*, section of a telium or teleutosorus of a true rust with a single enlarged teliospore; *G*, section of cluster cups or æcia and pycnia (above) with each spore fruit enlarged; *H*, conidiophores and conidia of the apple-scab fungus; *I*, three types of coremia; *J*, a habit sketch of pycnidia; *K*, section of a pycnidium with a spore tendril protruding from the ostiole; *L*, section of a stromatic pycnidium; *M*, section of a stroma with several immersed pychidia; *N*, detail of small portion of the wall of a pycnidium showing conidiospores and conidia or pycnospores; *O–R*, types of acervuli; *S–U*, types of sporodochia; *V*, three different types of conidiophores from spore fruits.

FIG. 25.—Semidiagrammatic drawings of ascigerous fruits and simple ascus-forming fungi. *A*, surface and sectional view of a closed ascocarp of Aspergillus, with a single enlarged ascus; *B*, habit sketch, surface view with escaping asci, enlarged ascus and section of a perithecium of a powdery mildew; *C*, vertical section of a typical ostiolate perithecium; *D* and *E*, sections of stromata with immersed perithecia; *F*, habit sketch of typical perithecia; *G*, section of a perithecium with long neck showing how the asci are released and forced up to the ostiole for the discharge of spores; *H*, section of a perithecium showing the manner of elongation of asci through the ostiole for the expulsion of the ascospores; *I*, two types of asci, one with terminal sphincter, the other terminal canal, structures used in spore discharge; *J*, four types of apothecia or fruits of cup fungi; *K*, section of a sessile apothecium; *L*, section of a stalked or stipitate apothecium; *M*, asci and paraphyses, or sterile filaments from a typical apothecium, showing one empty ascus with the lid separated; *N*, *O*, vegetative yeast cells and sporulating cells or simple asci; *P*, asci of a simple filamentous fungus, Endomyces; *Q*, leaf of peach affected by leaf curl, or *Taphrina deformans*; *R*, asci from the surface of the same leaf.

Fig. 26.—Semidiagrammatic drawings of basidium fruits. *A*, cranberry affected with rose bloom (*Exobasidium oxycocci*), showing enlarged flowerlike lateral shoot; *B*, basidia from the surface of one of the hypertrophied leaves shown in *A*; *C*, red leaf spot of the cranberry, with leaf and stem lesions which produce basidia similar to those shown in

cuplike receptacles; (7) a *pycnidium* (pycnid, for short), a more or less globular structure, generally embedded in the substratum and opening on the surface by a pore, or ostiole, through which the spores, *pycnospores*, are extruded, either in mass or in long coils or tendrils (spore horns); (8) an *acervulus* (pl. acervuli), typically a saucer-shaped, depressed structure which bears conidiophores over its exposed surface and sets the spores free by the rupture of the superficial cell layers of the host; (9) a *sporodochium* (pl. sporodochia) similar to an acervulus, but forming a stroma-like cushion, which breaks through the host tissue and bears conidiophores over most of its exposed surface.

The ascus-producing fruits are in marked contrast to the previous classes of fruits producing asexual spores (Nos. 1 to 9). In the leaf-curl fungi, the spore sacs form an extensive layer over the surface of some host part, without being grouped in definite fruiting bodies. Three general types of ascus fruits may be recognized: (1) the *cleistothecium*, a minute superficial, globular or slightly depressed black body, consisting of a surrounding wall completely enclosing one or more asci; (2) the *perithecium* (pl. perithecia), a more or less flask-shaped body, borne either immersed in host tissue or in a stroma of fungous tissue and enclosing a group of asci (sometimes also sterile filaments or *paraphyses*) which liberate the spores through a terminal opening in the perithecial wall by the forcible explosion of the ascus sacs; and (3) the *apothecium* (pl. apothecia), a disklike, saucer-shaped or cuplike body, seated on or in the substratum or raised on a short or long stipe or stalk and bearing the cylindrical asci, frequently mingled with sterile filaments or paraphyses, on the concave exposed surface. The apothecia vary in size from structures barely visible to the naked eye to others several inches in length or diameter, and are usually more or less fleshy in character. The spores are generally set free by the simultaneous explosion of a group of asci, producing the phenomenon known as "puffing of spores," in contrast to the behavior of perithecia, from which the spores are expelled by the explosion of the asci in succession.

Fig. *B; D*, base of stem showing a simple basidial felt (Hypochnus or Corticium type); *E*, hyphae and basidia from *D; F*, habit sketch and section of a plain resupinate sporophore (Stereum type); *G*, typical basidia with one sterile cell or cystidium; *H, I*, resupinate, shelving and stalked sporophores with toothed basidial surfaces (Hydnum type); *J*, sporophores of fairy clubs and coral fungi (Clavaria type); *K*, habit sketch of a resupinate poroid sporophore; *L*, sections of annual and perennial bracket sporophores (Polyporus and Fomes types); *M*, section through a single pore showing arrangement of the basidia; *N, O*, imbricated bracket fruits of the pore type; *P, Q*, stipitate or stalked sporophores, the basidia in pores; *R, S*, sporophores of gill fungi or toadstool forms (Agaricus type); *T*, section of a toadstool form through the middle of the cap or pileus and the stipe or stalk showing the varying lengths of the basidium-bearing plates or lamellae; *U*, arrangement of the gills as viewed from the under surface of the pileus; *V*, section of a portion of a gill showing arrangement of the basidia; *W*, upper surface of the sporophore of a common gill fungus (*Schizophyllum alneum*); *X*, under surface of the same sporophore showing the arrangement of the gills or lamellae.

In some of the more primitive basidium fungi there is no organization of a definite fruiting body, the basidia being produced from the general mycelium which develops on or in the substratum.   In the great majority of the basidium fungi the basidia are grouped and supported by rather complex structures which may be designated as *compound sporophores*. The various types are described in the introduction to Chap. XI.

CHAPTER VII

DISEASES DUE TO PHYCOMYCETES

In the classification of the fungi, three important groups or classes may be recognized:

✓ **1. Phycomycetes** characterized by a nonseptate mycelium and alga-like reproductive habits. ✓

✓ **2. Ascomycetes** characterized by a septate mycelium and the formation of spores (ascospores) in a specialized cell or *ascus*. ✓

✓ **3. Basidiomycetes** also characterized by a septate mycelium but with spores (basidiospores) formed *on* either a primitive or well-developed cell or *basidium*.

Each of the classes includes a large number of species, many of which are either obligate or facultative parasites and may be important causal agents of disease in plants. In the following chapters, consideration will be given to important diseases of our cultivated plants caused by representative species of the three above classes.

The fungi which form a nonseptate mycelium and reproduce by the development of either oöspores or zygospores, unless sexual spores are omitted, and by either swarm spores, conidia or other asexual types are included in the class of Phycomycetes, which may be conveniently divided into the two following subclasses:

A. The *Oömycetes,* in which the typical sexual reproduction is by the union of two unequal and dissimilar cells or gametes to form an *oöspore.*

B. The *Zygomycetes,* in which sexual reproduction is generally by the union of two equal and similar gametes or sex cells to form a *zygospore.*

OÖMYCETES

This subclass comprises the three following orders:

The chytrids (Chytridiales) with a plant body consisting mostly of a single unbranched or only slightly branched cell. This order includes many species parasitic only on algae, but also a number of important pathogens of our crop plants. The most important genera furnishing pathogens of seed plants are: *Plasmodiophora, Spongospora, Synchytrium, Physoderma* and *Urophlyctis.*

The water molds (Saprolegniales) with well-developed mycelium, but principally saprophytic in habit. Many species may be found in fresh waters of streams or lakes living upon dead insects, fish or plant

remains, while a few forms are parasitic on fresh-water algae, fish or other aquatic animals. Several destructive parasites of plants are to be found in the genus *Aphanomyces* of the Saprolegniaceae, the larger of the two families comprising the order.

✓ **The pythiaceous fungi, white rusts and downy mildews** (Peronosporales), which are principally parasitic in habit, and for the most part obligate parasites. The chief distinguishing features of this order are: (1) a well-developed, nonseptate mycelium which is intercellular with the exception of forms of Pythiaceae, which are also intracellular; (2) asexual reproduction by *conidia* (*zoosporangia*), which in the more primitive forms produce *swarm spores* and hence are sporangia, while in the highest types an infection hypha is the first product of germination; and (3) sexual reproduction, when this is not suppressed, by the union of unequal and dissimilar gametes to form *oöspores*. A large female cell or *oögonium* produces either a uninucleate or multinucleate gamete, while the male cell or *antheridium* is smaller and gives rise to several male gametes or sperm nuclei. *Fertilization* is accomplished by the union of the gametes of opposite sex. The three following families are recognized:

✓ *A. Pythiaceae,* without definitely differentiated conidiophores (*Pythium*) or with simple or branched sporangiophores (*Phytophthora* and *Trachysphaera*).

✓*B. Albuginaceae* or white rusts, characterized by: (*a*) the production of conidiophores (sporangiophores) in associated groups or *sori* beneath the host epidermis; (*b*) the unlimited production of conidia (sporangia) in chains; and (*c*) the germination of both conidia and oöspores by the formation of zoospores or swarm spores. Includes the single genus, *Albugo.*

✓*C. Peronosporaceae* or downy mildews, so called because of the white tufts or downy aggregates of aerial conidiophores formed on the surface of affected parts. The important features of the family are: (*a*) simple or branched conidiophores which generally emerge in small groups from stomatal openings; (*b*) the limited production of conidia (sporangia), a single spore being formed on each branch of the compound sporophores; (*c*) the germination of the conidia either by the formation of swarm spores or by direct development of infection threads; and (*d*) the germination of the oöspore by the direct production of a germ tube or infection thread. Includes the following genera: *Peronospora, Plasmopara, Peronoplasmopara, Sclerospora, Bremia.*

ZYGOMYCETES

Two orders are recognized:

The insect fungi (Entomophthorales) with asexual spores as conidia only, and sexual spores by the union of gametes of dissimilar size. One

species, *Completoria complens* Lohde is parasitic on the prothallia of ferns; most of the others are parasitic on insects.

The Black Molds (Mucorales).—The asexual spores are either conidia or sporangiospores, but the latter are the more common. Most of the species are saprophytes or weak parasites, but a few are obligate parasites of no importance to crop plants. In the seven families, only two genera, *Rhizopus* and *Choanephora*, are of importance as furnishing parasites of crop plants, the former producing only sporangiospores, the latter both sporangiospores and conidia. There is no morphological differentiation between male and female hyphae, but physiological differences exist. The uniting gametes may be formed on different branches of the same mycelium, the *homothallic* types, or they may be produced by separate mycelia, the *heterothallic* types. The heterothallic mycelia are different sexual types and may be designated as + or − strains. A + and − strain must be brought together to insure the formation of *zygospores*.

CLUBROOT OF CABBAGE AND OTHER CRUCIFERS

Plasmodiophora brassicae Wor.

The cabbage and other species of the mustard family are frequently attacked by a disease in which the effect upon the root system has suggested such names as "clubfoot," "clump foot," "clubroot," "clubbing," and "finger-and-toe disease."

FIG. 27.—Clubroot of cabbage, showing swollen and distorted roots and undeveloped head. (*After Cunningham, Vt. Bul.* **185**.)

Since the first intensive and authoritative study by Woronin (1873–1878) in Russia, the disease has become world-wide and attention has been given to it in the various countries, with many important contributions from Europe, Australia and America.

Symptoms and Effects.—The disease may affect seedlings, which after three to five weeks show retarded growth or a pale-green or yellowish color and roots ten to twelve times the diameter of normal ones. Seedlings that are infected early are usually killed by midseason, while later attacks are less serious, although the swollen distorted roots begin to decay towards the end of the growing season.

Six general types of hypertrophy have been recognized: (1) complete clubbing of main and lateral roots—*Brassica oleracea;* (2) clubs on main roots, laterals free—*Sisymbrium altissimum;* (3) clubs on lateral roots, main root free—*S. officinale* and *Erysimum cheiranthoides;* (4) clubs on main and lateral roots with club-free rootlets above the diseased portions —*Lepidium sativum;* (5) clubs as tumors of the root—*Raphanus sativus;* and (6) dark, decomposing spots on the root—*R. sativus.*

The hypertrophied roots are very frequently somewhat elongated or fusiform, and are well characterized by the descriptive name of finger-and-toe disease, while, in other cases (turnips or rutabagas), the swellings are very frequently globular and grouped mainly on the laterals, with the fleshy taproot more nearly normal, but sometimes turnips may present only a group of branched hypertrophied roots. (See Root Knot for hypertrophy caused by nematodes.)

Etiology.—Clubroot is caused by *Plasmodiophora brassicae* Wor., for many years classed as a slime mold but more recently grouped with the chytrids. Bacteria occur in the diseased tissue but are not the primary invaders, since they follow the pathogen and by their activities bring about the disintegration or rotting of the diseased roots, thus only contributing to the injury. The clubroot organism cannot be grown in pure cultures but successful inoculations have repeatedly been made by the use of soil contaminated with the spores, or of soil filtrates containing a suspension of the spores and more recently by bringing together cabbage seedlings grown under aseptic conditions and spores obtained directly from the interior of young clubs. This behavior, with the constant occurrence of plasmodia in the cells of affected roots, is sufficient proof of the active pathogenicity of *P. brassicae* Wor.

The spores which are set free by the decay and disintegration of the diseased roots may germinate at once or after a period of rest and may cause infections at temperatures from 16 to 21°C., although the optimum for germination is 27 to 30°C. The spores are spherical or slightly hexagonal and somewhat variable in size. Measurements recorded by different workers range from 1.6 to 4.3 μ with an average of 3.3 to 3.9 μ. In the process of germination the spore swells to be one-third larger, bulges on one side, the cell wall ruptures and the content escapes as a naked, uninucleate mass of protoplasm. The details of form and behavior of these myxamoebae or swarm spores as given by different observers are somewhat at variance. Three types have been described: (1) long spindle-shaped form, variable, uniciliate but also forming pseudopodia (Woronin); (2) pyriform, uniciliate, never amoeboid (Chupp); and (3) amoeboid and devoid of cilia (Honig). These different descriptions may be explained by the ability of the zoospores to change form.

The manner in which these myxamoebae or swarm spores bring about infection has been one of the disputed points in the etiology of the disease. Various observers have found amoebae or plasmodia containing two to six nuclei in the young infected tissue, but the exact behavior between the uninucleate, free, swarm-spore stage and the several-nucleate plasmodium within the host cells seems to be somewhat uncertain. According to one of the most recent reports the swarm spores first penetrate root hairs and form a small plasmodium with up to 30 nuclei, each of which organizes a zoosporangium. These then germinate, forming four to six small zoospores (gametes) which then migrate to the

FIG. 28.—*A*, cross section of a root badly infected with *Plasmodiophora brassicae;* *B*, cross section of a young healthy cabbage root; *C*, plasmodium passing through the end of a cell in the region of the cambium. (*A and B after Woronin; C, after Kunkel, Jour. Agr. Res.* **14,** *Plate* 70*I*.)

epidermal and cortical cells of the root and there fuse in pairs. The resulting zygotes give rise to plasmodia of typical clubs. No evidence of fusion of zoospores was found by Wellman (1930). Whether the infecting amoebae are uninucleate or become several-nucleate before penetration into the host cells is relatively unimportant.

The idea had generally prevailed that infection could take place only through young tissue of roots, but it has been shown that "old tissues are readily penetrated by the parasite." Successful infections have been secured from inoculations made near the root tips, at points far back from the regions producing the root hairs and even on rather old cabbage stems. When infection takes place through young roots, it is possible that the amoebae may penetrate through root hairs and other epidermal

cells of the primary cortex and pass on into the deeper tissues. However, it seems probable that this method of penetration is not common but rather that infection takes place through portions of roots from which the primary cortex has already been lost.

There are two methods by which the plasmodia may be spread throughout the affected root: (1) by the division of cells in which two or more amoebae are present; and, (2) by the migration of plasmodia from cell to cell. It has been shown that migration is more important than cell

Fig. 29.—Stages in spore formation in the plasmodium. The collection of the cytoplasm around the nuclei is shown in *B* and *E*; large vacuoles are shown in *C*; *D* shows the edge of a plasmodium about ready to form spores; *A*, plasmodium showing nuclear divisions preceding spore formation. (*After Lutman, Vt. Agr. Exp. Sta. Bul.* **176**.)

division. By increase in size and division of a plasmodium, a single cell may contain a considerable number of plasmodia. The amoebae penetrate into deeper lying cells passing from one cell to another, at the same time migrating somewhat along the longitudinal axes of the root, finally reaching the cambium. They also penetrate into cells on the inner face of the cambium but migrate more rapidly in the cambium and thus spread the infection lengthwise from the original center of infection. The plasmodia then migrate from the cambium into adjacent tissues of cortex or medullary rays. Carefully prepared sections will show plasmodia passing through the separating cell walls of adjacent cells. As a result of the presence of the parasite, certain cells are greatly increased

in size, and cell division is stimulated, the stimulus extending beyond the cells actually occupied by the parasite. In this way, a swollen or distorted root is produced. A single, spindle-shaped club is to be considered as a morphological unit resulting from a single infection that occurred at some point near its middle, rather than as a result of multiple infections.

Finally, some cells remain permanently infected, that is, the plasmodia do not migrate from them. These may be single, in longitudinal chains or in groups called *Krankheitsherde*. Affected cells may be crowded full of plasmodia which are highly granular from the presence of reserve food material in the form of oil globules. In the formation of spores, the plasmodia become filled with large and small vacuoles, the cytoplasm collects around the nuclei and the whole mass is thus cut up into uninucleate masses which round off and surround themselves with protecting walls.

A single diseased root thus produces millions of spores which are set free into the soil by its decay, and these are capable of infecting succeeding crops on the same ground or the disease may be spread to new fields. Wind dissemination or the migration of the motile swarm spores in the soil are minor factors in the spread of the disease. It may, however, be spread by soil carried on farm implements, on the feet of animals, by earthworms, through the use of contaminated fertilizer, by drainage water flowing from contaminated soils to healthy fields or by the use of infected seedlings.

Host Relations.—Extensive tests of susceptibility were carried out by Cunningham (1914), using over 100 species of which 11 remained immune, while the others showed varying degrees of susceptibility. Rochlin (1933) found that 14 of 50 wild and cultivated species remained free from infection. Practically all of our cultivated species show a susceptibility to the disease: varieties of cabbage, cauliflower, and brussels sprouts (*Brassica oleracea*), turnip (*B. campestris*), rutabaga (*B. rapa*), rape (*B. napus*), various mustards (*B. spp.*), alyssum (*Alyssum spp.*) and various other less frequently cultivated species. Naoumoff (1926) has reported the results of infections on 180 species in 49 genera, including the first report on species of the subfamily Thelypodieae. Jamalainen (1936) has tested 88 wild and cultivated crucifers and found 69 susceptible, 29 of which had not previously been recorded as liable to clubroot.

Of 13 varieties of cabbage tested by Cunningham in 1911, the four showing the most resistance were: Hollander, 26.5 per cent; Stone Mason, 14.4 per cent; Large Late Flat Dutch, 9.9 per cent; and Early Jersey Wakefield, 9.4 per cent free from clubs when grown on heavily contaminated soil as contrasted with 100 per cent infection for the most susceptible varieties. Thirteen varieties of radish tested in 1912 and 1913 showed that the percentage of clubbing varied from 1.8 per cent in

Early Scarlet Turnip to 92.3 per cent in Early Long Scarlet. Turnips and rutabagas tested showed that Sweet German, White Swede, Early White Milan, Early Snowball and Purple Top Aberdeen were relatively resistant, while Southern Curled, Early Purple Top Strap-leaved and Improved Purple Top Strap-leaved were relatively susceptible.

It is claimed that there is a direct relationship of resistance to infection to the quantity of glucosides which, on fermentation with myrosin, produce highly pungent mustard oils. Sinigrin is one of these glucosides, and the suggestion is made that control may be obtained by crossing to obtain forms rich in active glucosides.

Biologic strains have been reported: (1) a kohlrabi-cauliflower strain; (2) a radish strain; and (3) one affecting Savoy cabbage.

Predisposing Factors.—Clubroot is especially favored by acid soils. Spore germination and infection are not exclusively dependent upon the hydrogen-ion concentration, but no infection will ordinarily result above pH 7.2 to 7.8. The excessive use of acid fertilizers or highly nitrogenous manures and the withdrawal of lime by the action of smoke gases in industrial centers may be expected to favor the development of clubroot.

Spores germinate at a temperature range of 6 to 27°C. with the maximum germination at 25°C. Clubroot development occurs from 12 to 27°C., with the optimum from 18 to 25°C.

The disease does not develop where the moisture content of the soil is down to 45 to 50 per cent of its water-holding capacity but will occur when the moisture content is higher, reaching heavier infection as saturation is approached. Low-lying, poorly drained soil might then be expected to favor clubroot, and well-drained soils to inhibit it. Fewer infections with low soil moisture are thought to be due in part to a more xerophytic type of host structure. Exposure for 18 hours to favorable moisture relations may result in infection, consequently, heavy, prolonged rains may offer conditions for infection even in the best-drained soils.

Preventive or Control Measures.—Attention should be given to:

1. *Sanitary Practices.*—These are designed to prevent the contamination of new areas: (a) Diseased roots if fed to livestock should be thoroughly boiled before feeding; (b) guard against the transport of soil (on cultivators, horses' feet, etc.) from infected fields to new areas; (c) sterilize seedbeds, especially if clubroot is known to be prevalent in the environment. Corrosive sublimate, 1 ounce to 10 gallons of water, applied five times to badly infested seedbeds has given excellent protection, while more recent tests of acidulated mercuric chloride have been even better. Good results from the use of Uspulun and some other fungicides have been reported but are, in general, less satisfactory than with mercuric chloride. Control has also been obtained by watering with a 10 per cent solution of washing soda. Chlorinated nitrobenzenes under

the trade names of "Folosan" and "Brassisan" have been tested by Smieton (1939) and in field trials were nearly as effective as mercuric chloride.

2. *Crop Rotation.*—Continuous cropping to cabbage or other suscepts may lead to a heavy soil contamination by the clubroot organism, and, since it has been shown that the pathogen can live in a soil for three or more (six) years, a comparatively long rotation should be adopted in handling contaminated soils. Four or five, and preferably six, years should intervene between cabbage crops, and no cruciferous crop of any kind should be grown in the interim. Attention should also be given to the elimination of all weeds belonging to the mustard family.

3. *Use of Fertilizers.*—Barnyard fertilizer should be applied during the season preceding a susceptible crop, but not to a cabbage or turnip crop itself. The same rule would apply if acid phosphate is to be used as a fertilizer. Since most soils in which clubroot becomes severe are distinctly acid, the *use of lime* has long been practiced with marked success. Raw-ground limestone, caustic lime, air-slaked lime and hydrated lime applied several months before setting have been used, the last giving the best results for field use, at 1500 to 2000 pounds per acre. An increase in yield of cabbage from 672 pounds per acre to 23,082 pounds per acre has been reported from liming the soil. Calcium cyanamide, which is also of value as a fertilizer, has been recommended as a substitute for lime and is about twice as effective, pound per pound. In recent studies, on the effect of the nutrition of the host, Pryor (1940) reports that "the percentage of susceptible plants having clubs was in general increased slightly over that in the complete solution by an abundance of potassium, more by an abundance of nitrogen and most by the absence of sulfur or nitrogen."

4. *Drainage.*—As a supplement to other practices low-lying or water-logged soils, whether acid or nonacid, should be thoroughly drained, and the physical condition improved as much as possible.

5. *Resistant Strains.*—Breeding for resistant strains has shown some promise in turnips and swedes (Olsson, 1940).

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POWDERY SCAB OF POTATOES

Spongospora subterranea (Wallr.) John.

Powdery scab is a disease that attacks roots, stems, stolons and tubers, producing small hyperplastic galls on the first three structures and small, circular, scablike lesions or, more rarely, cankers on the tubers. Various common names have been applied to the disease, such as corky end, corky scab, powdery scab, Spongospora scab and potato canker, but powdery scab has been most generally used, especially in America. The name is descriptive of the character of the lesions and serves to distinguish the trouble from the more widespread and common scab (*Actinomyces scabies*).

It is the belief that the disease was introduced into Europe from Peru, South America, where it was endemic. It was first described as occurring in Germany in 1841 and has since become known in Great Britain and throughout northern Europe. It reached Canada in 1913, Maine in 1913 and 1914, and since then sporadic occurrences have been reported in other northern states from Maine to Washington and Oregon. More recently the disease has appeared in Australia, New Zealand, Tasmania and Kenya Colony and has been slowly extending throughout the U.S.S.R., where it has been reported as serious since 1935.

Symptoms and Effects.—Lesions of three different types are produced: (1) white galls on roots, stolons and stems resembling somewhat bacterial nodules, varying in size with some as large as garden peas; (2) typical powdery lesions or sori on the tubers; and (3) the *cankerous stage* in which there is an actual destruction of some of the flesh of the tuber, leaving hollowed-out eroded areas. The lesions of the first type cause but little effect on growth, while those of the third type are very severe, although less frequent than the typical powdery lesions which cause most of the damage to the tubers.

The typical tuber lesions appear at first on young tubers as small, slightly raised pimples or swellings with a slight discoloration of the surface. These swellings enlarge, become somewhat more raised and finally break down, leaving a cavity filled with a mass of brownish powder surrounded by the lighter colored, frayed-out periderm or skin.

These scab spots are usually circular or oval, $\frac{1}{16}$ to $\frac{1}{4}$ inch in diameter, may occur singly or in groups, in patches or well scattered over the surface, and may even coalesce in groups and thus obscure their normal form. The circular form, the ragged margins of the periderm and the central powdery mass are characteristic features. The typical form and character of the lesions may be modified by friction in handling so that tubers in storage may have the top and powder rubbed away, leaving only slightly depressed, empty lesions. Some lesions may not have matured sufficiently to rupture the periderm, but in many such cases

Fig. 30.—Powdery scab of potato showing characteristic appearance of open sori. (*After Morse, Maine Bul.* **227.**)

the spore powder may be recognized by breaking the external covering. In severe attacks, especially in moist soil, roundish or irregular, light- or dark-brown warts several times larger than the pustules may sometimes be formed, most frequently toward the terminal or seed end of the tuber.

The injury from powdery scab varies with the type of the attack. In mild cases, it consists only of surface scabbing, which disfigures the tubers and depreciates the market value of the crop, with but little reduction of real value as table stock. The seed value of affected stock is greatly lowered, unless it is to be grown in areas climatically unsuited to the development of the disease. In such regions, severely infected seed has given a good yield of clean tubers. In more severe cases of the common type, as well as the canker stage, there may be heavy losses, beginning in the field and extending into storage. Some fields in Maine have shown as high as 90 per cent of the hills affected, while 50 to 75 per cent was recorded for others. In certain areas in the Soviet Republic when heavy rains occur in May and June powdery scab is reported to cause 30 per cent reduction in yield as well as an increased liability to late blight.

The entrance of wound parasites may cause destructive types of powdery-scab dry rot, since the bottoms of the scab pits are protected by but little or no wound cork, so that penetration by fungi is relatively easy. Among the wound parasites, the most common form in Maine was

FIG. 31.—Section through a powdery-scab sorus showing disintegrated tissue and numerous spore balls. (*After Melhus et al., Jour. Agr. Res.* 7, *Plate* 12A.)

described as a new species, *Phoma tuberosa*. Other rot-producing organisms, especially bacteria and various species of Fusarium, may enter the Phoma lesions and assist in destruction.

FIG. 32.—Mature spore balls in an enlarged host cell. (*After Osborn.*)

Etiology.—The powdery scab is caused by *Spongospora subterranea* (Wallr.) John., now generally classed as one of the chytrids. The pathogen was first believed to be a slime mold and was later believed to be one of the smut fungi.

The powdery content of the scab sori consists of numerous, minute brown bodies known as spore balls, which are in reality collections of spores held together in more or less spongelike, spherical or ovoid masses, varying in size but averaging about 50 μ. According to Blattny (1935), the spore balls from roots, underground stems or stolons are only 48.5 μ in size, those of the tubers 52.1 μ, and are considered as varieties, the former as *radicicola*, the later as *tubericola*. Under favorable conditions these cells of the ball (all or part of them) may germinate. Each cell of the spore ball may produce a single, small, hyaline, uninucleate amoeba which generally escapes through an opening in the spore wall, leaving the spore ball intact. In other cases the entire spore ball disintegrates, setting free as many amoebae as there were cells in the spore balls. These amoebae are actively motile and soon crawl away from the spore ball from which they were produced. Under dry conditions they round up and may become surrounded by a thick, rough wall, or become encysted, in which state they are resistant to adverse conditions. These cysts may persist in the soil from year to year and with the return of favorable conditions may germinate and set free the amoebae again.

Numerous amoebae external to the host coalesce to form a plasmodium which becomes the infecting body, and this passes down through the epidermis, the lenticels and more rarely wounds constituting the principal infection courts, rather than the unbroken skin of the tuber. After penetrating the epidermis the plasmodium spreads out and forms a disk-shaped mass below the uplifted epidermis and the sound tissue beneath. Projections of pseudopodia begin to extend downward and push between the cells of the sound tissue. Small strands of protoplasm, the "infecting pseudopodia," are pushed through the softened walls of the host cells and in some manner become separated from the remainder of the plasmodium. Shortly after the cells become infected, they enlarge and elongate radially to five tö ten times normal size, forming giant cells, which are responsible for the raised condition of young lesions. Ultimately the giant cells are cut up into smaller cells, which are all infected. Finally each nucleus of the intracellular, multinucleate plasmodium organizes a spore, and these become grouped to form the characteristic spore balls. There will then be left in the sorus a group of spore balls, mingled with the fragments of old cell walls of host cells or other remains of host cells. Spore balls in the base of the sorus may germinate, form more plasmodia, which kill the invaded cells at once, deepen the lesion by these secondary invasions and form still more spore balls.

According to Ledingham (1935), zoosporangia may be formed in the root hairs and in root epidermis, which discharge biciliate zoospores.

Fusion of these is suggested by the occurrence of some binucleate, four-ciliate cells.

The source of an infection may be due (1) to the planting of affected tubers or (2) to planting clean tubers in a previously contaminated soil. The spore balls carried by infected seed tubers germinate in the soil under the same conditions as the seed, and plasmodia are produced which may cause the infections in the manner already described. Spore balls generally remain dormant during the winter, but in the spring many will germinate. Under favorable conditions, the plasmodia may lead a saprophytic life; under conditions of stress they may become encysted and so prolong the life of the parasite. It is uncertain how long the

Fig. 33.—A semidiagrammatic drawing of a section through a very young sorus, showing the infecting plasmodium as it pushes down between the cells. (*After Kunkel, Jour. Agr. Res. 4, Plate 29, Fig. 3.*)

pathogen may live in the soil, but some authorities believe that some infections will result after a lapse of three to five years.

Predisposing Factors.—The conditions for infection seem to be rainfall periods during the young stages of tuber formation, followed by cool, damp, cloudy weather. The proper temperatures coupled with poor soil drainage may give heavy infection. In America these favorable relations are formed mainly near the Canadian boundary and farther to the north. It is worthy of note that heavily infected potatoes planted at 15 different places on the Atlantic Coast from Massachusetts to Florida, and at six different points in Washington from Everett southward, yielded an absolutely clean crop. An ideal soil for infection is said to be one with large pore spaces, a high humus content, a high methyl-pentosan content and a large water-holding capacity. High infections have resulted in soil with 60 to 90 per cent moisture and a pH ranging from 4.7 to 5.9 (Naoumoff, 1936).

Host Relations.—In addition to the potato, other species of Solanum are susceptible to powdery scab. According to Melhus, of 16 species of Solanaceae planted in contaminated soil, seven developed infections, and one of these was the tomato. In no case were mature spore balls produced in any of the root galls. *Solanum nigrum,* a common weed, was not infected. Eight varieties and species of Solanum from South America were reported to be immune by Dorojkin (1937).

There seem to be marked differences in the susceptibility of different potato varieties, but it is uncertain if any are absolutely immune. Tests in Maine and in Russia have shown one or more varieties that remained free from infection, while one Russian worker (Dorojkin, 1937) reports that no commercial varieties were immune, but that Jubel, Cobbler and Parnassia were only weakly susceptible.

Control Practices.—The following control practices may be emphasized:

1. In regions climatically favorable for the disease use only seed free from the disease, selecting the most resistant varieties.

2. Avoid contaminated land for at least 3 to 5 years after an infected crop.

3. If contaminated land must be used give attention to careful drainage. Applications of sulphur up to 900 pounds per acre will reduce infections. Avoid lime as it will favor infection.

4. If infected seed stock or stock suspected of being infected must be used disinfection is recommended. The following have given the best results: (*a*) hot formaldehyde, 2 pints to 30 gallons of water at 46 to 50°C. for 5 minutes; (*b*) hot mercuric chloride, 4 ounces to 15 gallons of water at 44 to 45°C. for 5 minutes; or (*c*) meranin, a liquid organic mercury, 1 to 2000 for 20 to 30 minutes. The latter fungicide has been reported by Russian workers to be more effective than mercuric chloride.

5. Guard against the introduction or spread of the disease by attention to all possible sanitary practices. Spore balls may be carried by contaminated bags or other articles that have come in contact with infected tubers or with contaminated soils. Farm implements or contaminated manure may harbor the organism. Infected tubers or parings may be boiled and fed to hogs. Quarantines of infested areas may be established.

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BROWN SPOT OF CORN

Physoderma zeae-maydis Shaw

This is a disease of corn affecting both stalks and leaves and causing spotting or blight and lodging. It has been known in this country only since 1912, but has been called by a variety of common names, such as "corn measles," "corn pox," "dropsy" and "spot disease." The terms "rust" and "frenching" have been incorrectly applied to the trouble. The region of greatest prevalence extends from the northern boundaries of Tennessee and North Carolina southward to the Gulf, but the disease occurs in less severe form as far north as South Dakota and as far east as Delaware. It is also known to occur in China, Japan and other oriental countries.

Symptoms and Effects.—The brown spot attacks the leaves, both blades and sheaths, and also the stalks, but is rarely seen on the outer husks of the ears. In moderate infection the lesions are sometimes most evident between the fourth node and the ground, but in very severe infections leaf blades and sheaths of the entire plant may be affected and die prematurely, leaving a weakened and dwarfed plant.

FIG. 34.—Portion of a leaf blade of corn showing the effects of a severe attack by *Phys.derma zeae-maydis* Shaw. (*After Tisdale, Jour. Agr. Res.* **16** 1919.)

The first evidence of the disease is the appearance of slightly bleached or yellowish spots, 1 millimeter or slightly more in diameter, which soon become darker, and finally turn brown or reddish brown, with a lighter margin. Adjacent spots may coalesce and make more extensive areas. The spots on the midrib and leaf sheath are large, up to 5 millimeters in diameter, irregular in shape or almost square, and generally darker

than the leaf-blade lesions. In a severe infection they may cause the entire leaf sheath to become brown, owing to the death of the host cells and the accumulation of the brown sporangia. A reddening of the host cells is an accompanying effect, and may sometimes mask or obscure the exact extent of the lesions.

Toward the maturing period of the host, the epidermis over the lesions dries and becomes loose and broken, thus exposing the brown sporangial dust, which is readily liberated. Severe infections on the leaf sheath may kill the leaf before the plant reaches maturity. The lower nodes of a culm may be completely girdled by the fungus and so weakened that they break over before they are completely mature. In severe attacks of the disease, the spotting and death of the leaves and the lodging of the stalks all contribute to the loss. Based on reduction in yield of grain, the most severe cases have resulted in losses up to 10 per cent, with a material reduction in the forage value of the stalks.

Etiology.—Brown spot is caused by one of the Chytridiales, which was described by Shaw as *Physoderma zeae-maydis*. The disease has been produced artificially by spraying healthy plants with a suspension of the zoosporangia. The brown dust that is set free in the older lesions consists of large numbers of separated sporelike bodies, which behave as sporangia. They are 18 to 24 by 20 to 30 μ, with a thick, smooth, brown wall, slightly flattened on one side, which is provided with a circular cap or lid. The sporangia pass the winter in the dead remains of infected plants or in the soil and germinate as soon as mature or the following season or even later. In artificial tests they have remained viable for at least three years. They may remain as a residual contamination or be carried away by such agencies as insects, running water or wind and by various agricultural practices.

Under proper conditions of moisture and temperature, the sporangium germinates. There is an increase in size by the absorption of water, the lid or cap opens in a doorlike fashion, and the swarm spores organized within the escaping mass (the endosporangium) finally break through an apical papilla and swim away. Each swarm spore is 3 to 4 by 5 to 7 μ, provided with one long polar cilium and a large central oil globule. After a period of activity, a swarm spore settles down, loses its cilium, becomes slightly amoeboid and then germinates by the production of fine fibrous hyphae.

If the germination takes place on the surface of a susceptible host, one or more hyphae may penetrate the epidermal wall and then expand within the host cells to form special, enlarged vegetative cells called "Sammelzellen." These groups of enlarged cells (two or more) are always intracellular and give rise to other slender fibers which give rise at once to other enlarged cells or pass into adjacent cells and there

produce other groups of "Sammelzellen." The zoosporangia are formed directly from some of the enlarged cells or at the end of special hyphae which grow out from them. When sporangial formation is complete, the mycelium has entirely disappeared and the sporangia appear to fill the dead host cells in which they were formed.

Fig. 35.—A life-cycle diagram of *Physoderma zeae-maydis*. *A*, sporangium; *B*, *C*, opening sporangia showing early stages of zoospore formation; *D*, zoospores or swarm spores; *E*, germinating swarm spores; *F*, successive stages of infection with the development of large cells or "Sammelzellen" and connecting fibers; *G*, host cells filled with mature sporangia. The contents of host cells have been omitted. (*Adapted from Tisdale, Jour. Agr. Res.* **16**, 1919.)

Predisposing Factors.—When sporangia are present in a field and suitable moisture and temperature conditions prevail when the corn plants are not more than half-grown, the disease is likely to develop in severe form. The temperature factor is the most important and probably limits the severity of the disease in its northern range and excludes it from other cooler regions. The zoosporangia require a minimum temperature of 23°C. for germination, and the optimum temperature seems to be about 28 to 29°C., a temperature that would be uncommon for night conditions in much of the corn belt. The spread of the disease westward is probably limited by the semiarid conditions that prevail through much of the growing period. High temperatures and abundant and frequent rains through the early growth of the corn crop furnish ideal conditions and explain the range of greatest severity of the disease.

Low wet lands or lands near water are favorable to the disease, while higher well-drained lands are less favorable, especially in seasons of

moderate rainfall. At higher mountain elevations in the South the disease may be excluded by the cool summer nights.

Control.—Our knowledge of control measures is very imperfect, but certain practices that have a bearing on the development of the disease may be noted:

1. Since the most severe cases have appeared on land cropped to corn for a number of years in succession, and the sporangia are known to persist in the soil, crop rotation is dictated. The new corn field should be located as far as possible from the old field that produced a diseased crop.

2. The removal of the plants from the field as early and as completely as possible would do much to lessen the supply of infective material for the following season. If an infected crop is fed as stover or fodder, the barnyard manure should not be used to fertilize land which is to be planted to corn.

Varieties of dent, flint, sweet and pod corn have shown fluctuating degrees of susceptibility with no definite indications of resistance, although selection for disease resistance may still be possible.

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DAMPING-OFF

Pythium debaryanum Hesse

The death of young seedlings from the attacks of soil-inhabiting fungi is of frequent occurrence, but *Pythium debaryanum* is one of the most frequent offenders. It was first studied in Germany by Hesse in 1874 and De Bary in 1881, while our first important American contribution was by Atkinson in 1895. Since these early studies, it has been recognized as of world-wide prevalence.

Symptoms and Effects.—In the early onset of damping-off, the hypocotyl of the seedling may show a pale color due to the destruction of the chlorophyll; the invaded tissue assumes a dirty white color, shrivels or becomes constricted just above the ground line and the young seedling suddenly falls over since the stem is no longer able to support the cotyledons. The rapidity of onset of the trouble may well be expressed by saying that the seedlings "drop dead." This damping-off is of special importance in many kinds of seedlings grown in pots, flats or benches in the greenhouse or in many plantings in garden or field beds and also in some more extensive field plantings. Under varying

conditions, the losses range from the death of a few seedlings to the loss of a very high percentage of a planting. In very early and severe attacks some young seedlings may be killed before they ever reach the surface of the soil (preemergence damping-off).

It is a well-known fact that cuttings of herbaceous plants may be invaded by soil fungi and be prevented from rooting or be killed after they have rooted. *Pythium debaryanum* is one of the known offenders and has been studied as the cause of a blackleg or stem rot of geranium cuttings and a similar disease of carnation cuttings.

A water soft rot of potato tubers known as "leak" also is caused by *Pythium debaryanum*, and also a similar soft rot of dahlia roots.

Etiology.—This common cause of damping-off, *Pythium debaryanum* Hesse, produces a colorless, nonseptate, much-branched mycelium. Branches from this mycelium may penetrate the epidermis of the hypocotyl of a seedling and then spread through the cortical parenchyma growing both in and between the cells, until all of the tissues except the wood cells are invaded. The internal mycelium may spread even into the cotyledons, and, in some cases, primary infections may take place through the cotyledons.

Asexual reproduction occurs by the formation of *conidia* and *zoospores* while sexual reproduction is by the union of unequal gametes to form *oöspores:*

Asexual Reproduction.—The ends of the much-branched mycelium may form globular enlargements which are separated off by a cross wall. When these filaments grow up into the air, the enlarged end cells function as conidia and may develop swarm spores or germinate, after a resting period, with the formation of a hypha. Other terminal enlargements, especially if immersed, becomes sporangia at once with the formation of a lateral neck which develops a terminal globular enlargement. The entire contents of the sporangium is passed into this vesicle, and then divides to form the zoospores which are set free. These zoospores are oval, with two laterally attached cilia and, after a period of activity, come to rest, round off and form an infection hypha. If in contact with a susceptible host part, penetration of the epidermis may take place with the establishment of a new infection. Intercalary resting spores (chlamydospores) also may be formed, which germinate after the death of the supporting hyphae.

Sexual Reproduction.—Certain of the globular enlargements resembling the conidia or sporangia become *oögonia*. At the same time a cylindrical branch from below or from an adjacent hypha grows up by the side of the oögonium, comes in contact, expands somewhat and is separated off by a cross wall to form the *antheridium*. This then sends a fertilizing tube into the oögonium, and the nucleus of the antheridium

moves in and fuses with the nucleus of the oögonium. The egg cell then surrounds itself with a double cell wall and becomes an *oöspore*, which after a resting period may germinate directly to form an infection hypha.

The incidence of the disease especially on seedlings is favored by: (1) an abundance of moisture in the surface layer of the soil; (2) compact, poorly aerated surface soil; (3) thick seeding causing heavy stands; (4) poor illumination; and (5) temperatures between 20 to 30°C.

Host Relations.—No attempt will be made to enumerate the hosts affected by damping-off. *Pythium debaryanum* is one of the very important causes of damping-off of many species of ornamental plants and garden vegetables especially in hotbeds, cold frames and in the greenhouses; of deciduous and coniferous seedlings in open field seedbeds; and of certain field crops planted directly in the field. Many of our common weeds also are susceptible.

Other Causes of Damping-off.—In addition to *Pythium debaryanum* various other species of soil-inhabiting fungi may be responsible for damping-off of seedlings. Prominent among these may be mentioned species of the following genera: Aphanomyces,

Fig. 36.—*Pythium debaryanum.* *A*, branched mycelium; *B*, a young zoosporangium; *C*, zoosporangium with extruded contents which has organized swarm spores; *D*, free swarm spores; *E*, antheridium; *F*, oögonium; *G*, oöspore; *H*, an intercalary zoosporangium. (*Adapted from Sorauer.*)

Rhizoctonia, Sclerotium, Phytophthora, Thielaviopsis, Botrytis, Sclerotinia, Phoma, Volutella, Gloeosporium, Colletotrichum, and some species of Pythium other than *P. debaryanum.* Very severe infestations with parasitic nematodes may also produce results scarcely distinguishable from damping-off caused by fungi.

Control.—Damping-off may be prevented or greatly reduced by giving attention to certain cultural practices and either to soil sterilization or to seed disinfection.

1. *Cultural Practices.*—(*a*) By method of seeding and watering. Plant on the surface of moist soil and cover with a thin layer of sand, or if the sand is not used, plant in soil moist enough to ensure germination, cover the desired depth but do not water until seedlings have emerged. After emergence of seedlings, water on a rising temperature preferably in the morning. (*b*) Plant in pure sand that has been washed in hot

water (160°F.) and supplied with a nutrient solution. (*c*) Germinate the seeds in sphagnum moss and transplant. (*d*) For broad-leafed seedlings, plant following a cereal crop, rather than following a legume.

2. Soil Sterilization or Seed Disinfection.—This may be accomplished by the use of either heat or chemicals. (*a*) Heat sterilization by baking soil in an oven, by drenching with or immersing pots of soil in water at 208°F., by one of the methods of electric heat sterilization, or by steam sterilization. (*b*) Chemical treatments, the most important of which are liquid formaldehyde applied to the soil, formaldehyde dust mixed with the soil, red copper oxide dust treatment of seed (amount according to size of seed), watering the soil after planting with a mixture of copper carbonate (1 ounce) in water (6 quarts), watering flats immediately after seeding and also after emergence of seedlings with Cheshunt mixture or use Semesan for either seed or soil treatment according to manufacturer's directions.

A detailed consideration of seed treatments for the control of damping-off and related diseases of vegetables and other crops is presented by Haskell and Doolittle (1940).

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LATE BLIGHT AND ROT OF THE POTATO

Phytophthora infestans (Mont.) DeBy.

This is undoubtedly the most serious of all the potato diseases when conditions are favorable for its development. It was introduced almost simultaneously into Europe and North America sometime between 1830 and 1840, and by 1845 became epiphytotic in both Europe and eastern North America. The notable Irish famine of 1845 and 1846 was caused largely by the failure of the potato crop. The earlier views as to the parasitic nature of the disease culminated in the masterly work of De Bary in 1861–1863. The causal fungus was thought to be a native of the northern Andes from which section it was carried to North America, Europe and other countries, but more recently evidence has been presented (Reddick, 1939) that the fungus is enphytotic in Mexico, from which country it was carried on some plants of the nightshade family cultivated for ornament or for medicinal properties. It has now become world-wide, although it is limited in the different countries by climatic factors.

Symptoms and Effects.—The disease attacks the tops causing a *blight* and may also invade the tubers and cause a *dry or wet rot.*

Dead areas appear at the tips or margins of the leaves and spread downward killing the entire leaf in one to four days if temperature and moisture conditions are favorable, while in dry clear weather the number of leaf lesions is limited, and they remain small and dry up without involving the entire foliage. Severely blighted leaves curl and shrivel when they dry out, or, if they remain moist, they soon decay, often emitting an offensive odor. In warm, muggy weather the disease advances very rapidly and the entire tops may become blackened and wilted, followed by a wet rot involving the stems. Under humid conditions, a delicate whitish or grayish sporulating growth of fungous fructifications develop from the under surface, by pushing to the surface through the stomata. This aerial growth may be scanty or even absent under dry, sunshiny conditions.

Development of the disease in the tubers is responsible for either a *dry or a wet rot,* depending on the conditions which prevail. Under the least favorable conditions for the development of the disease, the tubers may show only superficial, slightly sunken, brown or purplish-black discolorations which penetrate only $\frac{1}{8}$ to $\frac{1}{4}$ inch and the affected portions remain relatively firm. This dry rot may be confined to a few small lesions or become extended to involve quite large areas. These dry-rot spots may be evident at digging time, but they frequently become more pronounced during the early portion of the storage period. Under favorable conditions for the progress of the rot, the tubers may be com-

pletely decayed before harvest, as a result of the combined action of the blight fungus and secondary invasions of bacteria or other fungi, giving the wet-rot phase of the disease.

Both foliage and tuber phases of the disease may be present and well developed, or either one may be inconspicuous and the other quite pronounced. The losses from the disease may be: (1) reduced yields due to few and smaller tubers as a result of the foliage attacks; (2) dry-rot tubers culled out at digging time; (3) the development of the disease after

Fig. 37.—Leaf of Irish potato showing terminal and marginal lesions of late blight. (*From N. Y. (Cornell) Agr. Exp. Sta. Bul.* **140**.)

storage; or (4) complete loss of the crop previous to digging time. The amount of injury from the disease may be inferred when it is noted that in regions where the disease is prevalent, its efficient control has resulted in yield increases of 40 to 233 bushels per acre.

Etiology.—The late blight is caused by *Phytophthora infestans* (Mont.) DeBy., a fungus originally described as *Botrytis infestans*. The fungus was at first considered only as a saprophytic accompaniment of certain physiological disturbances, but several early workers proved its parasitism, while the most complete and thorough treatise on the disease was published by DeBary in Germany in 1863. Since that time the disease has been a favorite subject for study in the regions in which it flourishes, and the output seems destined to continue.

The fungus develops an internal, intercellular, nonseptate mycelium in the affected parts and absorbs its food by means of variously formed haustoria which penetrate the cells. Slender, aerial, sparsely branched, septate hyphae, the *conidiophores*, grow out through the stomata in groups of one to five, and produce ovoid or lemon-shaped, multinucleate (7 to 30) conidia, 22 to 32 by 16 to 24 μ, which are at first terminal but become lateral as the parent branch continues its growth.

Ever since the true nature of the parasite has been recognized, speculations and disputes have been prevalent as to the occurrence of sexually formed *oöspores*. It is now known that antheridia and oögonia are formed which give rise to resting spores, or oöspores. These have been found in cultures and on plants and tubers in the field.

The conidia germinate in two ways: (1) by sending out a germ tube or infection thread; or (2) by the division of the contents to form biciliate

Fig. 38.—Sections through tubers affected with late-blight rot due to *Phytophthora infestans.*

swarm spores, which escape through a terminal pore and after a period of activity settle down and produce infection threads (see Fig. 40). The type of germination is influenced by temperature, moisture and substratum.

Infection hyphae produced either from the conidia or from the swarm spores may enter the leaves or stems through the stomata or even penetrate the unbroken epidermis. In tuber infections the entrance is probably through the lenticels and not from extension of the mycelium down the stem. The conidia are relatively short lived, remaining viable for only a few weeks, hence they cannot carry the fungus over the winter.

Various theories have been advanced to explain the yearly occurrence of the blight, but it has been definitely shown that the mycelium from infected seed pieces may grow up in the young shoots and sporulate on them after they reach the surface. Spores from these primary infections may start infections upon new foliage and the spread continues. It has

recently been reported that the fungus may remain alive for at least one vegetative season in diseased tubers left in the ground and can sporulate on remnants brought to the surface in cultivation (Naoumoff, 1939).

FIG. 39.—Diagrammatic representation of a square cut from a potato leaf infested with *Phytophthora infestans*; showing the fungus emerging through the stomata and the successive stages in the development of conidiophores and conidia. (*Redrawn from Vt. Bul.* **168,** *by Jones, Giddings and Lutman.*)

Since it is now conceded that oöspores are formed in nature, there is a possibility that the fungus overwinters in that stage also. Fields which contain no primary infections may become diseased by the transport of spores from neighboring fields by wind or insects.

The tuber lesions are produced by spores from blighted tops which are washed down into the soil, or from direct infection by mycelium in the soil, and some infections may result from contact with freshly blighted tops during digging time, while there may be some spread from infected tubers to sound ones, either in the soil or in storage, if sufficient moisture is present.

Fig. 40.—Stages in the germination of a conidium (zoosporangium) of *Phytophthora infestans*, and the germination of a swarm spore to form an infection thread. (*After Ward.*)

Climatic Relations.—It has been shown that neither the age of the plants nor the conditions of light, humidity or temperature operating on them increase or decrease their susceptibility to late blight, and consequently are not factors which influence the occurrence of epiphytotics. The various investigations have shown that the temperature and humidity of the environment acting upon the pathogen are the factors of most importance in contributing to the severe development of the disease.

In order to ensure infection, in addition to the presence of viable conidia there must be an accompaniment of the favorable moisture and temperature conditions. This requisite may be lacking since sporangia lose their ability to germinate very rapidly (1 to 2 hours in very dry air, 3 to 6 hours in air of 50 to 80 per cent relative humidity). Fluctuations in relative atmospheric humidity are more detrimental to germination than changes of temperature. The requisites for an epiphytotic are then a supply of viable sporangia, optimum temperatures for swarm-spore production and an ample supply of moisture, with the continuation of these conditions until infection has occurred. Sporangia will form in from 6 to 12 hours, depending on the temperature and humidity, and will produce swarm spores in ½ to 2 hours (10 to 25°C.), with penetration of host tissues by the germ tubes in a few hours (2 to 2½ hours

at 10 to 25°C.). After infection the lesions develop most rapidly at 18 to 21°C., but are checked by continuous temperatures above 30°. Three to five days later, if favorable temperatures continue, the lesions will be sporulating, and the process may be continued.

Physiological Forms.—Some recent studies have failed to show any morphological or physiological differences in a comparative study of isolations from California and New York. Müller, in Germany (1933), has recognized three biologic strains: type *A*, attacking all commercial varieties but not *W* strains; type *S*, attacking commercial varieties and *W* strains but not *Solanum demissum;* and transitional types. Later (1935) two biotypes were isolated from tomato. Reddick (1940) reports four races of late blight fungus in America. It is now claimed that breeding for resistance is complicated by the existence of physiological strains.

Host Relations and Varietal Resistance.—In addition to the potato, the late blight attacks various other wild and cultivated species of the nightshade family (Solanaceae) and has been reported on two species of figworts (Scrophulariaceae). Tomatoes, peppers and eggplants are attacked as well as numerous other less important species. As a blight and fruit rot of tomato, the disease has caused heavy losses in certain regions, especially the Virginias and southern California. It is reported that the fungus may be carried in and on tomato seed.

Commercial varieties of potatoes have shown varying degrees of resistance according to the following groups: (1) highly resistant; (2) moderately resistant; (3) intermediate; (4) moderately susceptible; and (5) very susceptible. More recently it has been claimed that susceptibility of foliage and susceptibility of tubers are not correlated. It is generally agreed that resistance is based on some substances in the tissues which retard or inhibit the growth of the parasite, but some workers have not admitted any change of susceptibility as the plants become older, if held under uniform conditions.

It is claimed that susceptibility is modified by certain environmental factors: (1) the water-nitrogen ratio, increased nitrogen affording resistance; (2) water content of the leaves, succulence favoring infection; (3) soil moisture, dry soils increasing resistance; and (4) supply of potash, a shortage lessening the infections, in this case, by a fungous rather than a host response. It is also claimed that tuber susceptibility is higher on clay soils than on sandy soils, owing to open or unsuberized lenticels on the former. Thickness of cell walls has been mentioned as a factor affecting tuber resistance.

Breeding for resistance has been in progress especially in America and Germany and promising hybrids have been produced, especially by using *Solanum demissum,* an immune species, as the pollen parent. Immune

or highly resistant hybrids, approximating to commercial types, have been developed—at least six in Germany (Müller) and about a dozen in America (Reddick). Sebago, a cross between Chippewa and Katahdin (Stevenson *et al.*), has shown moderate resistance to late blight and high resistance to mild mosaic. Several new blight-resistant varieties have been recently developed in Germany. These include Erika, Fruhnudel and Robusta (Snell, 1941).

 Control.—Late blight can be effectively controlled by spraying, and some excellent results have been obtained by dusting, but attention should also be given to other measures:

 1. *Selection of Seed.*—Careful inspection at cutting time to eliminate affected tubers or avoidance of seed stock from fields known to have been affected.

 2. *Storage at Low Temperatures.*—At 40°F. or under, the advance of the rot is very slow, while moisture and higher temperatures favor its spread.

 3. *Miscellaneous Practices.*—The incidence of tuber rot may be reduced by some of the following: (*a*) delay of digging until a week or more after the death of the tops; (*b*) high ridge culture to bury the tubers; (*c*) spraying the surface of the soil with Bordeaux or copper sulphate; (*d*) the cutting and destruction of tops in late attacks on unsprayed fields; (*e*) spraying the matured tops with $12\frac{1}{2}$ per cent sulphuric acid or 5 per cent copper sulphate; (*f*) especially for seed purposes, dig when the tops are green, dip in formaldehyde (1–99) or $CuSO_4$ and caustic soda (4–1–40) and store.

 4. *Spraying or Dusting.*—Two to six applications of Bordeaux, beginning when the plants are 6 to 8 inches high, using strengths varying from 4–4–50 to 8–8–50, the stronger formulas for the later applications. In light attacks two to three sprayings may suffice, but, under epiphytotic conditions, six applications or even more may be necessary. Burgundy mixture has also given good control. Independent of the control of late blight, spraying has given increased yields owing to control of insects and the lessening of physiological defects. It has recently been shown (Skaptason, 1940) that the accumulation of copper in the soil from continued spraying with Bordeaux has greatly reduced tuber infection.

 Varying results have been obtained with copper-lime dust (dehydrated $CuSO_4$, hydrated lime and calcium arsenate), some workers claiming as good or better protection than with Bordeaux, the majority, however, reporting poorer control, but the difference in yield of sprayed and dusted plots has not been great.

 In regions of frequent occurrence or great severity of the disease, spraying or dusting is accepted as a routine practice. In some regions

meteorological offices issue warnings to growers as to the time when weather conditions are favorable for an outbreak, so that the applications of spray may be made at the right time. ✓

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THE WHITE RUST OF CRUCIFERS

Albugo candida (Pers.) Kuntze

This is the most common species of the white rusts which attack various cruciferous plants throughout the world.

FIG. 41.—Habit sketch of leaves of salsify affected with white rust (*Albugo tragopogonis*), showing sori similar in external appearance to those of *A. candida*.

Symptoms and Effects.—Prominent white blisters varying in size and shape, the *sori*, that later rupture and become powdery may appear on any host parts except the roots.

In many cases the affected leaves are not greatly modified. The spore pustules may appear on one leaf surface only or on both surfaces with gradations from a few localized infections to complete invasion, and the amount of injury will depend upon the severity of infection. On some hosts, the leaves from infected stems may be "thickened, fleshy, pallid and distorted or inrolled" and, in severe infections, more or less reduced in size.

Invaded stems frequently show localized or extended swellings or enlargements, which may be slight or very pronounced, and exhibit sharp bends, turns or even complete spirals. A proliferation from lateral buds that are normally dormant may result in a bushy growth.

The entire inflorescence may be invaded or various flowers or flower parts may be affected, causing distortion of the axes, suppression of flower development or discoloration and malformation of flower parts. The following are some of the possible modifications: (1) floral organs swollen and fleshy and green or violet in color instead of normal; (2) failure of petals and stamens to fall; (3) a reversion from the cylic to the strobilate type of flower.

When examined by transmitted light or when cut across, the affected organs may sometimes appear darkened towards the end of the growing season owing to the internal development of large numbers of brown-walled spores.

When the white rust occurs alone, it may cause little or no injury or it may prove more serious, especially if associated with the downy mildew (*Peronospora parasitica*). Seedlings may be dwarfed or killed outright under favorable conditions for the disease. Affected flowers may be sterile and so injure crops grown for seed.

Etiology and the Causal Organism.—The white rust of crucifers is an obligate parasite and when studied by early botanists was thought to belong to the true rusts, hence the common name. In all of the earlier American literature the fungus appears as *Cystopus candidus* Lev., but more recently as *Albugo candida* (Pers.) Kuntze.

First infection results in the development of a copious growth of internal, intercellular, nonseptate mycelium, which forms globular haustoria within adjacent cells. Characteristic groups of conidiophores develop beneath the epidermis, raising it, to make whitish pustules or extended blisterlike areas, the sori. As soon as the covering epidermis ruptures, the conidia or summer spores (sporangia) are set free and may germinate at once, if favorable conditions are offered, and thus serve for the rapid dissemination of the fungus.

The conidiophores, 35 to 40 by 15 to 17 μ, are short, basally branched, club-shaped structures which give rise to simple chains of an indefinite number of spores formed by successively cutting off the terminal portion

by a cross wall. When mature the simple chains of multinucleate conidia are separated one from another by short necklike projections, but, finally, the older, terminal portions of the chains break, setting free the individual conidia which are easily disseminated by air currents.

Morphological specialization within the species has recently been recognized on the basis of conidial size: (1) the variety *microspora*,

Fig. 42.—*Albugo candida.* *A*, a hypha with growing tip *t* and haustoria *h* between the pith cells of *Lepidium sativum; B*, a group of conidiophores and conidia; *C–E*, stages in the formation of swarm spores from conidia; *F*, germination of a swarm spore that has come to rest; *G*, germinating swarm spores about to send infection hyphae into a stoma; *H*, an infection hypha of *Phytophthora infestans* penetrating the epidermal wall of potato stem. (*After De Bary.*)

14.5 by 15.5 μ, from Cardamine, Capsella, Draba and Arabis species; and the variety *macrospora*, 18 by 20 μ from Brassica and Raphanus species.

The conidia or sporangia are relatively short lived, their period of viability being limited to about six weeks after maturity, but they are able to germinate at once if they are afforded favorable conditions of moisture and temperature. After 2 to 10 hours' immersion in water a

conidium shows a segmentation of its contents into four to eight poly-
hedral masses, which separate and escape one by one to the outside or,
in adherent groups or the whole mass, may be discharged into a bladder-
like structure.　The swarm spores then take on their typical form of
ovate to kidney-shaped bodies, two unequal cilia appear from the flat-
tened or concave side and they swim away from the mother cell.　After a
period of activity they come to rest, absorb the cilia, form surrounding

Fig. 43.—*Albugo candida*.　*A*, mycelium with young oögonia *og*; *B*, oögonium *og*
with the egg cell or oösphere *os* and the antheridium *an*; *C*, a mature oögonium with a
fully developed oöspore *os*; *D*, optical section of oöspore shown in *C*; *E–G*, successive
stages in the germination of an oöspore with the formation of swarm spores.　(*After De
Bary*.)

cell walls and germinate to form infection threads which may enter the
host through stomata.　It was formerly believed that first infection
took place through cotyledons or seed leaves only, but it has been shown
that various parts of older plants may become infected if suitable con-
ditions are afforded.　An incubation period of seven to ten days is
required for susceptible species or longer for resistant species.　In
resistant species the germ tube may enter the stomatal chamber but make
little growth beyond the development of a haustorium.

Toward the end of the growing season oögonia and antheridia are formed on the internal intercellular mycelium, each appearing on separate but adjacent hyphae. The oögonium is a large globular multinucleate cell, the antheridium a smaller, more or less globular cell. One or more antheridia come to occupy a position close to an oögonium. The protoplast of the oögonium becomes differentiated into a peripheral or external multinucleate zone, the *periplasm*, and a central uninucleate mass, the *egg cell*, or oöplasm (Fig. 43).

The multinucleate antheridium produces a short, tubelike outgrowth, the *fertilizing tube*, which penetrates the periplasm and comes in contact

FIG. 44.—Fertilization in *Peronospora parasitica* (1) and *Albugo candida* (2, 3); *og*, young multinucleate oögonium; *an*, antheridium; *os*, uninucleate oösphere or egg; *a*, fertilizing tube of the antheridium which introduces the male nucleus; *o*, fertilized egg cell surrounded by the periplasm. (*After Wager.*)

with the egg. The antheridial or male nuclei are discharged through this tube into the egg cell and one male nucleus fuses with the female nucleus, thus bringing about the act of fertilization.

Following fertilization the periplasm is absorbed, and the fertilized egg cell develops a dark cell wall with characteristic external ridges, becomes filled with oily or fatty reserve food and, as a fully developed *oöspore*, lies within the old empty oögonial cell.

The oöspores will not germinate until they have been subjected to winter temperatures and are set free only by the weathering and decay of the host parts. Under favorable spring conditions, the oöspore wall splits and allows the internal, transparent, saclike membrane containing the swarm spores to protrude. This sac finally ruptures and the biciliate swarm spores which escape may produce new infections under favorable conditions.

There seem to be two types of infection: (1) a general or systemic, in which the whole plant is affected, resulting in a stunted growth and the appearance of the spore pustules on all parts; (2) a local infection, in which single leaves, stems or flower parts are directly invaded.

Predisposing Factors.—Temperature conditions affect not only the germination of the spores but also the apparent susceptibility of the host. The optimum germination temperature is close to 10°C., the minimum very close to freezing, while the maximum is about 25°C. Spore germination proceeds normally during the cool spring season when there is also an abundance of moisture, but in many regions the summer temperatures are sufficiently high to check or very greatly lessen germination. Experiments have shown that chilling of the host is an equally important factor in inducing infection, since chilled seedlings showed high infection (95 per cent) while the controls not chilled were low (5 to 15 per cent). A fall in temperature, which leads to the deposit of the dew, may provide the stimulus for spore germination, probably increases the susceptibility of the host and at the same time furnishes the medium in which the swarm spores develop. For this reason the greatest development of the white rusts is during the cool periods of early spring.

Host Relations.—The white rust of crucifers is found on many species of the mustard family throughout the world, both wild and economic plants being affected. The fungus is not confined to the Cruciferae but occurs on various species of Capparidaceae in Europe and in India.

The most important cultivated hosts for America are: cabbage, cauliflower, cress, mustards, horse-radish, radish, rutabaga, turnip, water cress, wallflower, and stocks. In addition there are over 40 wild hosts from 21 different genera. The most common weed hosts are shepherd's-purse (*Bursa bursa-pastoris*), peppergrass (*Lepidium virginicum*) and *Sisymbrium officinale*.

The existence of specialized races or biological species has been demonstrated. The form on the common radish has been shown to pass readily to other varieties of *Raphanus sativus* and also to *R. caudatus*, less frequently to white mustard (*Brassica alba*) and cabbage (*B. oleracea*), but failed entirely when inoculated on 10 other species. Another observer has reported the strain from turnips capable of infecting the cabbage and its derivatives. *Arabis alpina*, a weed, has furnished spores which infected nine other species belonging to six different genera, but this strain failed on radish, mustard and cabbage. Recent studies in Japan have revealed at least three physiological strains: one from radish affecting all radishes but no other species; another on Chinese mustard (*Brassica juncea*); and a third on *Brassica campestris chinensis*. The second and third strains were able to infect different groups of *Brassica* species. More recent studies in Japan have recognized at least

five biological forms on (1) Capsella; (2) Draba; (3) Arabis; (4) Raphanus; and (5) Brassica. Studies of collections from widely separated parts of Great Britain have shown the existence of at least 21 biologic forms all of which can infect *Brassica alba*.

✓**Prevention or Control.**—The white rust is not generally sufficiently severe to justify expensive control measures, but certain practices are of value: (1) crop rotation to avoid infection from a previously diseased crop; (2) clean culture to keep down all cruciferous weeds; (3) the destruction of infected crop refuse by burning to prevent carrying oöspores over the winter; (4) spraying, recommended only in the case of very severe attacks and for certain crops (horse-radish), is not satisfactory as the fungus overwinters in the roots. Lime-sulpher has been shown to be ineffective and Bordeaux to cause injury to horse-radish. (5) A promising line is the development of resistant varieties.

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DOWNY MILDEW OF GRAPE

Plasmopara viticola (B. and C.) Berl. and DeT.

This disease, which is endemic in the eastern United States, spread to France around 1874 and then a little later to other parts of Europe, where it became a serious disease because of frequent epiphytotics. It did not reach South Africa until 1907, Australia in 1917 and New Zealand in 1926 and is now prevalent in nearly all parts of the world where grapes are grown. It does not occur in the drier regions or those with prevailing high temperatures such as Palestine, Algeria, portions of South America, the arid southwest of the United States and all of the Pacific Coast region west of the Rockies, except for a single report on a wild species, *Vitis californica*. Since 1900, eleven epiphytotics have been recorded for the grape-producing areas of Europe. ✓The voluminous literature on this disease is largely from French and German workers. ′

✓ **Symptoms and Effects.**—The disease may affect leaves, leaf stalks and tendrils; young canes; flowers; and fruits at different stages of development.

Leaf infections show first upon the upper surface as pale-yellow spots of variable size and form which merge into the surrounding green tissue without any distinct line of demarcation. The early transparency of these spots has suggested the French name of "oil spots." Under

sufficiently humid atmospheric conditions, the lower surface of each spot is soon occupied by a conspicuous aerial, downy, milk-white coating (conidiophores and spores) which suggests the common name "downy mildew." Older spots become brown owing to the killing of the leaf tissue and are evident on both leaf surfaces. Under dry conditions and also on the old brown lesions, the aerial growth of the under surface is absent or scanty. Leaf spots may be few in number and then large,

Fig. 45.—Underside of a grape leaf showing groups of conidiophores of *Plasmopara viticola*.

or so numerous as to coalesce and involve nearly the entire leaf area or extended portions. In the more resistant varieties, the leaf spots are less conspicuous and in very resistant varieties "subinfections" may occur in which the invading hyphae perish together with some of the surrounding host cells.

Lesions on *young canes* appear water-soaked at first, then yellowish green and finally brown. In severe infections the young shoots may be dwarfed, twisted and deformed with only small leaves and, in the more extreme cases, killed.

Flowers may be completely blighted by early attacks or *fruits* may be attacked when young or when approaching maturity. If the berries are attacked when young, that is, when about $\frac{1}{4}$ inch or less in diameter, further growth is checked, the gray superficial growth of the fungus appears, the berry darkens and finally dries up. In berries attacked when nearly full grown, but before color appears, the fungus only rarely appears on the surface, but the infected berries darken owing to the death of the constituent cells. This change progresses slowly, dark patches appear at separated points, the skin becomes withered and wrinkled and finally the whole berry becomes shrunken and dark brown. The condition on the younger fruits with the copious growth of the mildew has sometimes been designated as "gray mold," while the characteristic changes of the older fruits have suggested the name of "brown rot."

The effects of the disease may be briefly summarized:

1. The loss of functional leaf tissue caused by heavy infections or shedding affects the fruit even though it is not directly invaded, causing abnormal ripening, with less juice and a lower sugar content, while the production for the following year may be seriously menaced.

2. Direct blossom attacks may cause a failure to set fruit, while later invasions may cause either mummification or rotting and shelling or dropping of the diseased berries.

Etiology.—The downy mildew, *Plasmopara viticola* (B. and C.) Berl. and DeT., forms a characteristic nonseptate, intercellular mycelium of thin-walled hyphae of irregular diameter in the affected tissues and there absorbs its food supply by numerous thin-walled, globular haustoria which are pushed into the cell cavities. After a period of mycelial development which has given rise to "oil spots," the hyphae become aggregated just beneath the stomata (lower leaf surface), and, if moisture conditions are favorable, three or more aerial hyphae grow out through the stomatal opening and form branched conidiophores bearing numerous conidia (sporangia) or summer spores (Fig. 46). These, massed together, give the downy spots on the lower leaf surface or cause the gray mold of fruits.

The globose, ovoid or more elongated conidia, 10 to 18 by 15 to 31 μ (maximum 40 to 50 μ), are borne singly on the sterigmata, or terminal tips of the conidiophore branches. When the detached conidia which are multinucleate are brought into favorable conditions of moisture and temperature, each nucleus with some adjacent protoplasm is organized into a swarm spore. These swarm spores escape from the sporangium through a terminal opening as planoconvex, biciliate, naked bodies which swim about in the moisture into which they are liberated. After a period of activity, they become more or less rounded, absorb their cilia, secrete a cell membrane and soon send out an infection thread or germ tube,

which may enter a stoma of any susceptible host part and establish a new infection.

Two types of conidiophores and conidia are formed: (1) the much-branched conidiophores with the ordinary conidia, which germinate by the formation of one to six swarm spores; and (2) groups of short unbranched conidiophores, each terminated by a large conidium (25 by

Fig. 46.—Downy mildew of the grape. *A*, conidiophores with conidia emerging through a stoma from an intercellular mycelium (represented as solid black); *B*, three stages in the formation of oöspores; *C*, stages in the germination of a conidium to form swarm spores. (*After Millardet.*)

35 or even 55 μ) which forms 8 to 10 swarm spores. The type appears to be influenced by host and environmental relations, with other forms intermediate between (1) and (2).

In the later summer or autumn, oögonia and antheridia, very similar to those described for *Albugo candida*, are formed within the diseased tissues of leaves (more rarely stems) and oöspores are matured. The oöspores are 25 to 35 μ in diameter, and form a thick, rough epispore. They remain dormant until spring, when they are set free by disintegration of host tissues and, if subjected to favorable conditions of temperature

and moisture, germinate by the production of a short unbranched pro-mycelium, which bears a single large conidium or sporangium. This conidium produces swarm spores in the customary manner and these, liberated on the surface of young leaves or splashed there by rains, originate the first lesions. These soon develop conidia, which are carried to new leaves or to other uninfected vines and thus spread the disease. Leaves can be infected as soon as stomata are well formed or when they are 2.5 to 3 centimeters long if moisture and temperatures are favorable, while the fruits may be infected until they are 13 millimeters in diameter, after which the stomata are closed. The conidiophores emerge from "oil spots" (lower surface) between one and three o'clock at night) and conidia from these produce infections after an incubation period of five or more days, depending on the temperature and air humidity.

Climatic Relations.—The occurrence and spread of downy mildew is influenced by temperature and air humidity. The oöspores germinate in the spring at temperatures of 11 to 32°C. (optimum 25°C.) and from April to June may induce primary infections from the swarm spores produced by the first sporangia (conidia). If not completely dried out, some oöspores may persist to the next season, and according to some investigators be responsible for the initiation of the disease after a year of apparent absence.

Secondary infections are produced during the growing season from the new crops of conidia disseminated by wind and rains so as to reach the under surface of leaves or other susceptible parts. The conidia germinate slowly at 2 to 3°C., after 2 to 3 hours at 10 to 12°C., after 40 to 45 minutes at 20 to 21°C., and not at all at 32°C. They remain viable for eight to ten days in 85 per cent moisture at medium temperatures, or several weeks at lower temperature. The temperatures necessary for infection are: 9° minimum, 18 to 24°C. optimum and 28 to 30°C. maximum and at 21 to 24°C. infection can take place in $1\frac{1}{2}$ to 2 hours. Young leaves may be infected at 70 to 85 per cent humidity, older leaves at 80 to 100 per cent, while the optimum is 95 to 100 per cent humidity. Primary infections require heavy rains, while secondary infections may occur with light rains, fogs or heavy dews even at 10 to 13°C., if the leaves remain wet for a few hours after midnight, and become more general with heavier rains. Hot and dry weather with plenty of sunshine may prevent established infections from fruiting, while rains and heavy dews with high air humidity are favorable if the temperature requirement is fulfilled.

Infection is also influenced by environmental factors operating upon the host: (1) to produce open stomata; (2) to produce a higher proportion of soluble carbohydrates, nitrogen and phosphorus. If soil and air are too dry, stomata remain closed, and, if the soluble food is too low, infection is retarded or lessened. It is claimed that susceptibility is influenced

by the calcium-potassium ratio in the leaves and fruit, and that, there-
fore, plants grown in soils rich in calcium are the least affected. Rainy
seasons affect the calcium-potassium ratio and thus increase susceptibility.
Host Relations and Variety Resistance.—The downy mildew of grape
will infect practically all wild and cultivated grapes and is known also on
woodbine (*Panthenocissus quinquefolia*) and Boston ivy (*P. tricuspidata*).
Vitis vinifera, the cultivated grape of Europe, is very susceptible, and the
native American varieties are more resistant. Many hybrids using suscep-
tible and resistant species have been produced, especially in France, to
combine resistance with desirable qualities, with the result that three
groups have been recognized: (1) resistant to both downy mildew and
powdery mildew, and spraying never necessary; (2) spraying necessary
in epiphytotic years only; and (3) yield and quality good with but few
treatments. The Concord, Niagara, Catawba and Delaware, varieties
very important in the Eastern United States, are derivatives of our native
wild species, and the latter, thought to be part *vinifera*, is more susceptible
than the others. Franklin is unaffected by either downy mildew or
black rot, and America, Dakota, Neosho and Suelter have shown only a
trace of each (Demaree *et al.*, 1938). It is claimed that the hybrids or
species which readily absorb calcium are resistant, while those which
take up very little calcium are highly susceptible.

Control.—In regions where downy mildew is a problem, main reliance
must be placed upon spraying, but other preventive practices should be
given attention: (1) the selection of resistant varieties or hybrids; (2) the
avoidance of practices which may increase the opportunity for infection,
such as: (a) close planting or dense stands; (b) planting in low, poorly
drained sites; (c) low trellis wires; (d) weed intercrops, as they hold the
moisture and retard drying; (e) permitting the growth of water sprouts;
or (f) cutting back when the stock shows infection; (3) selection of ferti-
lizers to lessen susceptibility; (4) the protection of bunches by cellophane
covers; and (5) the elimination of surface, oöspore-containing debris by
plowing or cultivating when conditions will permit.
     The time for spraying, and the number of applications will vary with
the varieties to be protected, the locality and the meterological factors:
(1) most resistant varieties, no spraying or only in epiphytotic years;
(2) susceptible varieties, from 2 to 17 applications depending on condi-
tions. The dates for spraying may be based on the stage of growth of
vines, the older method, or by the more exact "incubation-calendar"
method based on the time of maturity of spores. It has been pointed
out that the latter is reliable only in regions having a relatively dry
climate, because of the rapid succession of secondary infections in humid·
areas (Osterwalder, 1940). For moderate severity three applications
may suffice; (a) when shoots are 6 to 8 inches long; (b) just after blossom-

ing; and (c) before the fruit changes color, but, under very favorable conditions, at intervals corresponding to each 15 centimeters of branch elongation, or for each 25 to 30 centimeters elongation for conditions less favorable to infection. The "incubation-calendar" method has been used with marked success in Europe and, according to reports, has doubled the yield in Baden during the last 10 years. Control still continues to be a problem in many sections during years of epiphytotics. Bordeaux or Burgundy sprays have consistently given the best protection, but many other formulas and proprietary preparations have been tried with varying degrees of success. Bordeaux, 5-5-50 strength, suffices with moderate severity, but even up to 8-8-50 has been recommended for epiphytotic conditions. Since conidia or swarm spores fix the copper or absorb it until a toxic limit is reached, the more epiphytotic the disease, the stronger must be the spray to be effective. Failures in control are due mainly either to too weak sprays or to incorrect timing of the applications. A special colloidal copper spray (Bosc, 1933) has been recommended because of its adhesiveness, fungicidal efficiency and freedom from scorching. Copper-containing dusts have not afforded protection equal to the copper sprays. Recently a special spray has been recommended by Casale (1936) as equal to Bordeaux, but containing only one-tenth the amount of copper. Nickel sulphate has been shown to be equal to copper sulphate in efficiency (Arnaud, 1937). An electromotor spraying plant has recently been used at the Swiss Federal Station (Jenny and Huber, 1940).

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DOWNY MILDEW OF ONION

Peronospora schleideni Unger

This disease of onion in which the causal fungus invades both leaves and bulbs has been called onion blight, onion mold, white blight, white blast, or mildew, but is more properly designated as downy mildew, a more characteristic name. Following the first record of the disease in England by Berkeley in 1841 and by Trelease in America in 1884, numerous workers in various parts of the world have called attention to its serious nature. A very complete account of the disease was published in 1904 by Whetzel from studies made in New York, and a later publication from the same state by Cook (1932) lists 57 different reports from various parts of the world. Either mild or destructive attacks have been reported from the British Isles, the various countries of continental Europe, Australia and New Zealand, China and Japan, Bermuda and some other Atlantic islands. In the United States the disease is present in a large percentage of the states from Maine to the Mississippi Valley and is especially destructive in Washington, Oregon and California. The most frequent reports are from New York, Ohio, Louisiana, Oregon and California.

In addition to its occurrence on the common onion, downy mildew is known to occur on garlic, leek, shallots, Welch onions, potato or multiplier onion, Egyptian or tree onion and some of the wild species of Allium.

Symptoms and Effects.—The first evidence of the disease is the appearance of a purplish or violet surface growth of a downy or fuzzy nature a short distance back from the tips of the older leaves. The affected areas may turn yellow, then wither and thus cause the leaf to fall over. The same type of growth may cover the surface of the leaves in more extended areas and lead to a complete blighting of the tops. These blighted tops are soon overrun by a black mold of the Macrosporium type. It has been shown by Murphy and McKay (1926) that the mycelium under some conditions will spread down the leaves and reach the bulb plate, from which it may spread upward into other leaves not previously invaded. The general effect on market onions will be a reduction in size of the bulbs, depending on the severity of the foliage infection, in the heavier infestations resulting in bulbs too small for market. On onions grown for seed the fungous lesions may develop at any point on the seed stalk and sometimes even within the inflorescence. Some of the stalk lesions, especially if severe and located toward the base, may cause the stalk to fall over and prevent the maturing of the seed, while others in a more terminal position if less severe may permit the maturing of seed before girdling has been completed.

FIG. 47.—Downy mildew of onion. A second attack. Lower dried-up leaves killed by first attack. New growth, which was strong and vigorous, now affected. Leaves becoming weak, owing to collapse of tissues. (*After Whetzel.*)

Early attacks have been reported to destroy onions in the seedling stage, the attacks resembling damping-off. Attacks on older plants cause a reduction in size of bulbs, the reduction being especially marked in early infected plants. This is caused by the destruction of the leaves, when they should be manufacturing food to be supplied to the developing bulbs. Onion downy mildew also causes losses in storage, as infected bulbs may produce green shoots prematurely and rot, especially on the more susceptible varieties. It is also reported that the disease increases the percentage of thick-necked bulbs, which are especially liable to rot in storage.

Etiology.—Downy mildew of onions is caused by *Peronospora schleideni* Unger, which produces an internal, intercellular, nonseptate mycelium.

FIG. 48.—Mycelial threads of onion mildew between the large conductive cells of the leaf; *a*, a hypha with several haustoria; *b*, branched or coiled haustoria; *c*, branched haustorium wrapped about the nucleus. (*After Whetzel.*)

This mycelium spreads through the leaves and obtains its nourishment by the production of suckers or haustoria, specialized branches that penetrate the cells. These haustoria are either simple coiled hyphae, or they may be branched and sometimes enwrap the nucleus. The internal vegetative mycelium is able to develop two kinds of reproductive organs, first *conidia* and later the sexually produced *oöspores*.

Conidial or Asexual Stage.—Branches from the internal mycelium emerge through the stomata and give rise to branched aerial conidiophores 300 to 700 μ high on which each terminal may give rise to a conidium. These are the structures that give rise to the downy or fuzzy growth noted under the description of symptoms. The conidia are elliptical, measure 44 to 52 by 22 to 26 μ, and germinate by the formation of an infection hypha (rarely two) sometimes from one end, but more frequently from the side. New infections are produced by the penetration of the infection hyphae through the stomatal openings.

The optimum temperature for germination of fresh conidia has been reported as 11 to 15°C., with 3°C. as the minimum and with no germination at 27°C. or above. Conidia are relatively short lived, remaining viable for 15 to 17 hours in saturated air and only for a few hours (1½ to 2) in dry air. The most favorable conditions for the production of the conidiophores and conidia are high humidity (98 to 100 per cent) with some water on the surface of the leaves, in the form of raindrops or dew. The period of incubation from infection to the production of a new crop of conidia has been shown to vary from 11 to 15 days under temperature conditions approaching the optimum.

Sexual Stage.—Under favorable conditions the internal mycelium will develop the sexual organs: *oögonia* in the form of globular swellings, around 30 μ in diameter, generally located at the end of a hyphal branch; and *antheridia*, smaller cells of irregular, more flattened form which come to lie adjacent to the oögonia. Both oögonia and antheridia are cut off from the hyphae on which they are borne by the formation of cross walls. The oögonium soon differentiates a denser, central globular protoplasmic mass, the *oösphere* or egg cell. A short tube grows out from the face of the antheridium in contact with the oögonium, penetrates

Fig. 49.—Mature conidiophore (*a*) with mature conidia (*b*); conidia germinating (*c*); conidium (*d*) with germ tube entering a stoma. (*After Whetzel.*)

the latter and permits the migration of the antheridial or sperm nucleus into the oögonium, where fusion with the egg nucleus occurs, thus completing fertilization. The fertilized egg cell is then transformed into a pale-brown, thick-walled *oöspore* with a densely granular content and several large oil drops. The oöspores are rather resistant to extremes of moisture, cold and heat and thus may serve to carry the fungus over the winter. When released from decaying leaves in the spring, they may germinate and cause infections in the new crop.

It has been shown that there is an extensive air or wind dissemination of conidia, as revealed by the use of spore traps exposed on an airplane flying over infected fields. Conidia caught at a height of 1500 feet showed

germination up to 75 per cent. It has also been shown that these spores may "remain viable for several days at temperatures and humidities that often prevail in the spring for many days in the Northeastern States" (Newhall, 1938). It is also the belief that perennial topset and multiplier onions growing in home gardens may be a source of spores to spread the disease to adjacent commercial fields. In some localities, especially in Ireland, the oöspore stage is reported to be rare in some years but very abundant in others, as based on studies from 1931–1938 (McKay, 1939), as many as 1176 being found per square millimeter. It was also shown that the oöspores remained viable for five or more years, hence might survive in the compost heap.

Control.—No single practice will afford a complete control of downy mildew, but the various methods that have been recommended will be presented.

1. *Rotation of Crops.*—Since the winter spores are known to live in the soil and since a certain percentage retain their infective power for five or six years, it is advisable to practice a rotation with nonsusceptible crops, selecting those suited to the environment. In general practice a three- to four-year rotation may suffice, as the percentage of viable spores will be very small after that period.

2. *Sanitation and Cultivation.*—The exclusion of the disease from new land may be accomplished by using disease-free seed and sets, also by avoiding the introduction of transplants from infected soils, or of soil from infested fields on tools. Tops and culls which may be systematically infected should be collected and destroyed by burning, rather than being left to disintegrate in the soil. Under some conditions in California a good seed crop has been matured by pruning off the infected leaves, thus allowing the seed stalks to develop with but little infection.

3. *Selection and Regulation of Environment.*—Fields should be selected which have good air and soil drainage, as the disease is favored by excessive dew and fog and by high soil moisture, which afford conditions for sporulation and reinfection.

4. *Spraying.*—Control of mildew of onions by spraying with Bordeaux was recommended by Whetzel (1904) and some others during the earlier studies, but with varying degrees of success, using a 4–4–50 mixture. The waxy surface of the foliage plus the necessity for frequent applications has made control by spraying difficult and probably of doubtful commercial value. Some improvement has resulted from the use of spreaders and stickers such as rosin-fish-oil soap, casein, or Penetrol, but even with these improvements it seems that spraying is still "impractical, costly and of doubtful value" (Cook, 1932), although rather limited tests by Yarwood (1937) have shown a reduced infection under both

greenhouse and field conditions by spraying with Bordeaux plus Penetrol, lime-sulphur with sodium oleyl sulphate, and with rosin. Recently /McWhorter and Pryor (1937) have obtained some promising results with malachite green and certain combinations that are more toxic to downy mildew than any of the combinations of Bordeaux.

5. *Varietal Selection and Breeding for Resistance.*—Because of the practical difficulties of fungicidal control, the development of resistant varieties is highly desirable. Some progress has been made showing three sources of resistance: (a) a male-sterile selection from the Italian Red variety showing highly resistant foliage and immune seed stalks; (b) another selection of the Italian Red with only slightly resistant foliage but with seedstalk immunity (superior to type a); and (c) an F₁ hybrid between Red 21 and two inbred lines of Stockton Yellow Flat, showing seedstalk immunity. The ultimate aim is a combination of desirable type with complete immunity to both leaf and seed-stalk infections.

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RHIZOPUS DISEASES

Rhizopus nigricans Ehr.

This organism is responsible for the rotting of various fruits and vegetables and also attacks seed and seedlings on the germinator.

The Organism.—Two types of spores are produced: asexual spores in sporangia; and sexual spores or zygospores, according to the general type, for the Mucoraceae. The hyphae formed by the germination of the asexual or sporangiospores show a granular, richly vacuolated protoplasm, remain without cross walls and branch and rebranch until an interlacing tangle of mycelium is developed in the substratum. Under suitable conditions groups of erect, aerial fruiting hyphae will grow up from the substratum, while others (stolons) remain vegetative and bend over to form a cluster of branched rootlike hyphae which penetrate the substratum. Fruiting branches bearing spherical globular enlargements, the *sporangia*, grow up from this group. One or more stolons may grow out from the group, "strike root" and develop another group of sporangiophores, and this process may be repeated. The fungus thus becomes

differentiated into the vegetative or root hyphae, distributive hyphae, or stolons, and the aerial sporangiophores.

Asexual Spores.—As the sporangiophore reaches its full length, it swells out at the tip into a spherical body, the sporangium. Numerous

Fig. 50.—General habit of Rhizopus. *A*, root hyphae, which penetrate the substratum; *B*, aerial hyphae or sporangiophores bearing terminal sporangia *C*; *D*, a stolon. (*After Sinnott.*)

minute nuclei are scattered through both central and peripheral protoplasm, but they are more abundant in the peripheral portion. As growth continues, the denser peripheral portion becomes more sharply marked off from the central vacuolated portion, and, finally, a cell wall

Fig. 51.—Diagrams showing the method of the columella and spore formation in *Rhizopus nigricans*. *A*, a young sporangium; *B*, showing the dome-shaped layer of vacuoles outlining the columella; *C*, showing early stage of cleavage; *D*, showing the contraction stage; *E*, expansion or polyhedral stage. (*After Schwarze, Mycologia* 14, 1922.)

is laid down which separates the external *sporangium*, or sporogenous portion, from the central sterile portion, the *columella*. Surface furrows or clefts appear in the dense peripheral protoplasm, and these cut progressively inward, until the dense sporogenous protoplasm is cut up into

angular, two- to six-nucleate masses of variable sizes which soon round off to form the spores. During the process of maturing, the spores excrete a homogeneous slime which fills the intersporal spaces, and this, by swelling, may break the sporangium wall, but there is no explosive mechanism. The remnant of the sporangium wall may persist around the base of the columella as the so-called *collar* and separate the columella from the expanded end, or apophysis, of the supporting hypha. Some of the spores may remain for a time sticking to the columella, but they are ultimately scattered, leaving nothing but the old columella, which may finally become everted.

The sporangia are 100 to 350 μ broad, snowy white when young, but black when old. The spores are variable in size, subglobular or broadly oval, 6 to 17 μ in diameter, frequently with one or two blunt corners, and with the external pale-gray wall marked by fine lines.

Sexual Spores.—In the formation of sexual spores, two hyphae come in contact with each other, a papilla-like protrusion is formed from each at the point of contact, and these continue to grow in length to form the *progametangia*. Each progametangium cuts off an end cell, a *gametangium*, containing a *coenocytic gamete*. The cell walls of the two gametangia are dissolved at their point of contact, the two gametes fuse, the nuclei unite in pairs, and the product of the fusion surrounds itself with a thick brown wall to form the *zygospore*, supported by the enlarged remaining portions of the progametangia, now called the *suspensors*. A zygospore germinates by the formation of a hypha, which soon gives rise to one or more primary sporangia. A gamete that fails to unite with another one may sometimes become transformed into a spore very similar to a true zygospore and then is called an *azygospore*.

The uniting gametes are formed on separate and distinct mycelia, physiologically male and female, and the sex cells or gametes may be designated as plus (+) and minus (−). Fusion to form the zygospores occurs only when two separate strains (+ and −) are brought together on the same substratum. The mature zygospores are dark brown, globular or subglobular and 160 to 220 μ in diameter, with external wall covered with hemispheric warts or projections.

SOFT ROT OF THE SWEET POTATO

The soft rot of the sweet potato caused by *Rhizopus nigricans* is also called mush rot, vinegar rot or leak. In the earlier studies of sweet potato rots, it was reported that Rhizopus also caused a ring rot, but more recent investigations have shown that this is a distinct disease caused by *Pythium ultimum* Trow.

Symptoms and Effects.—Roots affected by *soft rot* become very soft and water-soaked, the rotted tissue finally becomes a cinnamon or choco-

late brown and a clear liquid oozes out if the rotted tissue is broken. Pressure in the bins causes the breaking of the skin and the watery fluid may leak out, making adjacent roots wet, thus indicating the presence of the trouble. Nothing is seen of the aerial sporangiophores of the fungus unless the rotting potatoes are exposed to a very moist atmosphere or are broken open, when the characteristic sporangiophores will frequently develop in large numbers. With the evaporation of moisture, the potato dries up, finally becomes mummified and, in this final stage, is frequently referred to as dry rot.

Soft rot is not confined to the harvested crop in storage but may appear in the field previous to digging time, and is most prevalent in low, moist areas, in delayed harvesting or when the roots have been

FIG. 52.—Strawberries slightly and severely attacked by *Rhizopus nigricans*, the cause of leak. Berries held for 48 hours in moist chambers.

injured in cutting the vines. Soft rot may also cause trouble to the sets in the hotbed, rotting the seed pieces and injuring the young sprouts.

It has been claimed that nearly 20 per cent of the estimated 30 per cent loss from storage rots of sweet potatoes can be attributed to the ravages of *Rhizopus nigricans* or other Rhizopus species which behave in the same way. The trouble still continues to take a heavy toll after the potatoes have been shipped to market.

Etiology.—It has been proved by pure-culture inoculations that *Rhizopus nigricans* Ehr., can cause the symptoms and effects described and that entrance is very largely through mechanical injuries of some kind. Infections are especially likely to occur during sweating, and poorly ventilated storage houses greatly favor the trouble, owing to the slow evaporation of the moisture. The rapidity of the advance of the rot varies with conditions, but, at room temperature, a potato may be completely rotted in four to six days.

The spores of *Rhizopus nigricans* may be found almost anywhere in dust and dirt, in the field and in the storage house. Some roots which were infected in the field may soon produce spores in the storage room, and, since these spores are resistant to desiccation, viable spores are ready to settle into any crack or bruise and start infections wherever sufficient moisture for their germination is available. The mycelium in the rotted roots is rather short lived (10 to 15 days), and is followed by various saprophytes in succession on the same substratum. *R.*

Fig. 53.—Development of sporangiophores from cut surface of an apple rotted by *Rhizopus nigricans.*

nigricans is the most common species of Rhizopus found on sweet potatoes, but eight other species are able to produce a similar rot. The different species can be roughly grouped into: (1) the high-temperature forms which thrive best at temperatures varying from 20 to 40°C.; (2) the intermediates, growing best at 20 to 35°C.; and (3) the low-temperature forms, making best growth at 15 to 20°C. *R. nigricans* belongs to the low-temperature group, and is more favored by the temperatures at which sweet potatoes are ordinarily held. The rot is caused by the secretion of the enzyme, pectinase, which dissolves the middle lamellae of the cells and so causes their separation.

Varietal Susceptibility.—On the basis of susceptibility to Rhizopus rot, sweet potatoes have been divided roughly into three groups based on

percentage of infection and rapidity of rotting: (1) *very susceptible*, including Gold Skin, Little Stem Jersey, Early Carolina, Florida, Red Brazil, Haiti, Yellow Belmont and Dooley (100 per cent); (2) *intermediate*, including Porto Rico, Big Stem Jersey, Triumph, Pierson, Florida and Dahomey; (3) *quite resistant*, Nancy Hall and Southern Queen.

Other Rhizopus Diseases.—A number of other rots caused by *Rhizopus nigricans* may be enumerated: (1) a leak or soft watery rot of strawberries, which is an important transportation trouble; (2) leak or melters of the Irish potato (caused also by Pythium); (3) a storage or transportation rot of pome fruits, stone fruits and small fruits; (4) a soft rot of tomatoes; (5) a tree rot of figs; and (6) a seedling infection of corn on the germinator.

Control.—Special care is required in harvesting, curing and storage. Important features of harvesting are: (1) dig only when roots are well matured; (2) avoid frost injury before or after digging; (3) dig in warm, dry weather rather than in wet periods; (4) do not store wet potatoes in great bulk; (5) avoid long exposure to hot sun; (6) dig only what can be dried and picked up before night; (7) dig and handle with extreme care to prevent cutting or bruising.

Curing for 10 days at a temperature of 80 to 85°F. at a relative humidity of 90 per cent, followed by storage at 55° (50 to 60°F.) has been recommended. Too low a humidity increases shriveling, while humidity above 85 per cent may favor infection.

The disinfection of seed roots with mercuric chloride is quite generally recommended. Dip the bushel hampers or wooden crates in a solution of mercuric chloride, 1 ounce to 8 gallons of water, for 10 minutes and then spread out to dry at once. After treating 10 bushels, add $\frac{2}{5}$ to $\frac{1}{2}$ ounce of mercuric chloride and restore solution to the original volume. Discard the solution after treating 50 bushels, as continuous use renders it ineffective.

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IMPORTANT DISEASES DUE TO PHYCOMYCETES

For key references on these diseases see F. D. Heald, "Manual of Plant Diseases," second edition, pp. 449–454; 489; 504.

CHYTRIDIALES

Principal host	Common name of disease	Scientific name of causal organism
Cabbage and other crucifers	Clubroot	*Plasmodiophora brassicae* Wor.
Potato	Powdery scab	*Spongospora subterranea* (Wallr.) John.
Cabbage	Seedling disease	*Olpidium brassicae* Wor.
Flax, etc	Blight	*Olpidiaster radicis* (deWild.) Pascher
Potato	Wart	*Synchytrium endobioticum* (Schilb.) Perc.
Cranberry, etc	Gall	*S. vaccinii* Thomas
Corn	Brown-spot disease	*Physoderma zeae-maydis* Shaw
Alfalfa	Crown wart	*Urophlyctis alfalfae* (Lagerh.) Mag.
Beet	Root tumor	*U. leproides* (Trabut) Mag.

SAPROLEGNIALES

Peas, etc	Root rot	*Aphanomyces euteiches* Drechsler
Sugar beet	Root blight	*A. cochlioides* Drechsler
Tomato	Root water mold	*A. cladogamus* Drechsler
Radish	Black root	*A. raphani* Kendrick
Tomato	Root water mold	*Plectospira myriandra* Drechsler
Sugar cane	Root water mold	*P. gemmifera* Drechsler

PERONOSPORALES (*Pythiaceae*)

Numerous	Damping-off and stem rot	*Pythium debaryanum* Hesse
Potato	Leak	*P. debaryanum* Hesse
Sugar beet and many others	Damping-off	*Pythium aphanidermatum* (Edson) Fitz.
Cucumber, beans, etc	Cottony leak	*P. aphanidermatum* (Edson) Fitz.
Citrus	Brown rot and gummosis	*Phytophthora citrophthora* (S. and S.) Leon.
Black walnut and deciduous fruit trees	Crown and trunk canker	*P. citrophthora* (S. and S.) Leon.
Potato	Late blight and rot	*P. infestans* (Mont.) DeBy.
Tomato	Blight	*P. infestans* (Mont.) DeBy.
Potato	Rot	*P. erythroseptica* Pethyr.
Tomato	Buckeye rot	*P. parasitica* Dastur
Citrus	Foot rot or *mal di gomma*	*P. parasitica* Dastur
Castor bean	Seedling and leaf blight	*P. parasitica* Dastur
Rhubarb	Crown or foot rot	*P. parasitica* Dastur
Lima bean	Downy mildew	*P. phaseoli* Thax.
Peony	Phytophthora blight	*P. cactorum* L. and C.
Numerous hosts	Damping-off, blight, crown rot, fruit rot, etc.	*P. cactorum* L. and C.
Lilac	Phytophthora disease	*P. syringae* Kleb.
Citrus	Australian brown rot	*P. syringae* Kleb.
Colocasia and caladium	Blight	*P. colocasiae* Rac.
Para rubber	Black thread and leaf fall	*P. palmivora* Butler
Cocoa	Pod rot and canker	*P. palmivora* Butler
Coconut	Bud rot	*P. palmivora* Butler
Tobacco	Black shank	*P. nicotianae* BdH.
Chestnut	Ink disease	*P. cambivora* (Petri) Buis.
Tomato, potato, etc	Foot rot	*P. cryptogea* P. and L.
Coffee and cocoa	Downy mildew	*Trachysphaera fructigena* T. and B.

IMPORTANT DISEASES DUE TO PHYCOMYCETES.—(*Continued*)

PERONOSPORALES (*Albuginaceae*)

Principal host	Common name of disease	Scientific name of causal organism
Cabbage and other crucifers..........	White rust	*Albugo candida* (Pers.) Rous.
Salsify and other composites.........	White rust	*A. tragopogonis* (Pers.) Rous.
'Sweet' potato' and other *Ipomoea spp*..	White rust	*A. ipomoeae-panduranae* (Schw.) Swingle
Amaranths.......................	White rust	*A. bliti* (Biv.) Kuntze
Purslanes.......................	White rust	*A. portulacae* (DC.) Kuntze

PERONOSPORALES (*Peronosporaceae*)

Millet, etc.......................	Green-ear disease	*Sclerospora graminicola* Schr.
Corn, wheat, rice and grasses.........	Downy mildew	*S. macrospora* Sacc.
Corn, teosinte and sorghum..........	Philippine downy mildew	*S. philippinensis* Weston
Corn, sugar cane and *Saccharum spontaneum*......................	Downy mildew	*S. spontanea* Weston
Corn and teosinte.................	Downy mildew	*S. maydis* (Rac.) Butler
Grape...........................	Downy mildew	*Plasmopara viticola* (B. and C.) Berl. and De T.
Sunflower and artichoke.............	Downy mildew	*P. halstedii* (Farl.) Berl. and De T.
Carrots, parsnips, parsley, etc........	Downy mildew of Umbelliferae	*P. nivea* (Ung.) Schr.
Cucurbits.......................	Downy mildew of cucurbits	*Peronoplasmopara cubensis* (B. and C.) Cl.
Hops............................	Downy mildew	*P. humuli* M. and T.
Lettuce, etc......................	Composite downy mildew	*Bremia lactucae* Regel
Cabbage and other crucifers..........	Downy mildew	*Peronospora parasitica* (Pers.) Tul.
Alfalfa and clover.................	Downy mildew	*P. trifoliorum* DeBy.
Spinach.........................	Downy mildew	*P. spinaciae* Laub.
Beet............................	Downy mildew	*P. schachtii* Fuck.
Pea.............................	Downy mildew	*P. pisi* (DeB.) Syd.
Pansy and violet..................	Downy mildew	*P. violae* (Schm.) DC.
Rose............................	Downy mildew	*P. sparsa* Berk.
Opium poppy and other *Papaver spp*..	Downy mildew	*P. arborescens* (Berk.) DeBy.
Tobacco.........................	Blue mold	*P. hyoscyami* DeBy.
Onion...........................	Blight or mold	*P. schleideni* Ung.

ZYGOMYCETES (*Mucorales*)

Sweet potato.....................	Soft rot	*Rhizopus nigricans* Ehr. and other Rhizopus spp.
Irish potato and fruits..............	Leak and rot	*R. nigricans* Ehr.
Lily.............................	Bulb rot	*R. necans* Massee
Cotton..........................	Boll rot	*R. nodosus*
Various vegetables.................	Vegetable rot	*R. fusiformis* D. and P.
Apples, pears, etc.................	Fruit decay	*Mucor pyriformis* Fisch. and other Mucor spp.
Squash..........................	Blossom blast and fruit rot	*Choanephora cucurbitarum* (B. and Rav.) Thax.
Hibiscus.........................	Blossom blast	*C. infundibulifera* (Carrey) Sacc.
Dahlia..........................	Blight	*C. spp.*
Peach...........................	Storage rot	*C. persicaria* Eddy
Tobacco.........................	Leaf mold	*Blakeslea trispora* Thax.

CHAPTER VIII

DISEASES DUE TO ASCOMYCETES

GENERAL CHARACTERS OF ASCOMYCETES

The Ascomycetes represent a class of fungi characterized by a septate mycelium and by the formation of spores (ascospores), typically 8 in number, but sometimes only 2 or as many as 16, *in* a specialized cell or *ascus*. The asci may be developed as an extensive layer of palisade-like cells on the surface of the host as in the leaf-curl fungi or they may be organized into definite fruiting bodies or *ascocarps*. Two general types of ascocarps are formed: (1) *apothecia* characterized by the formation of an extended layer of numerous *asci* or spore sacs, the *hymenium*, generally mingled with sterile hyphae, or *paraphyses*, arranged in a palisade-like layer with supporting or accessory parts; and (2) *perithecia*, globular or flask-shaped structures containing one to many *asci*, with or without sterile hyphae or paraphyses, either completely enclosed by the perithecial wall or opening to the exterior by a pore or *ostiole*. The perithecia may be simply saclike cavities immersed in an aggregate of fungous tissue, a *stroma*, or they may be provided with definite walls. In the latter case they may be single and embedded in the substratum, seated upon it, grouped in or on *stromata* or aggregates of fungous tissue that are either embedded in the substratum or are superficial (see Fig. 25).

A few of the ascomycetes produce no other spore stage than asco-spores, but, in by far the greater number, one or more forms of conidial fruits are developed. These conidial fruits generally constitute a very prominent stage in the life history of the parasitic species, making their appearance on the diseased parts, while the ascigerous stage may frequently be formed as a saprophyte on dead parts or even independent of the host. The three types of conidial fruits are: (1) Conidia formed on specialized branches or conidiophores that are single, fascicled or grouped into extensive layers or united to form *coremia* or *sporodochia;* (2) conidia on conidiophores grouped in an *acervulus;* and (3) conidia (pycnospores) formed by conidiophores contained within a pycnidium (see Fig. 24). The ascus stage is referred to as the *perfect* stage while the conidial stage is called the *imperfect* stage.

CLASSIFICATION OF ASCOMYCETES

The following is a brief tabulation of the orders of ascigerous fungi which furnish important plant pathogens:

1. Exoascales represented by the Exoascaceae containing the genus Taphrina including the *leaf-curl fungi*, in which the asci form an extended layer on the surface of diseased host parts without the organization of definite fruiting bodies.

2. Helvellales, mostly saprophytic fungi, with fleshy, waxy or gelatinous ascocarps usually differentiated into a sterile stalk bearing an expanded ascigerous portion, but including also a parasitic form (Rhizina) with stalkless or sessile fruits.

3. Pezizales, or cup fungi, with flat, saucer-, cup- or vase-shaped apothecia, either immersed or erumpent and sessile or stalked. The following genera furnish important plant pathogens: Monilinia, Dasyscypha, Pseudopeziza, Pyrenopeziza, Fabraea, Neofabraea and Cenangium.

4. Phacidiales, with leathery*or carbonous, depressed, globular or elongated apothecia, free or stromatic, and opening by rays or slits. The most important genera furnishing plant pathogens are: Keithia, Coccomyces, Phacidiella, Trochila, Rhabdocline and Rhytisma.

5. Hysteriales, with minute black, leathery or carbonous, typically elongated, boat-shaped apothecia opening by an elongated slit. The genera Lophodermium, Hypoderma and Hypodermella furnish parasitic species.

The four preceding orders (2 to 5) in which the ascus fruits are apothecia are frequently grouped as the *disk fungi*, or Discomycetes, while the following orders in which the ascus fruits are perithecia may be called the *sphere fungi* or Pyrenomycetes.

6. The Perisporiales and a few allied orders characterized by simple, spherical, tuber-shaped or stromatic ascocarps which are mostly nonostiolate. The following genera are of importance as furnishing either decay-producing forms or definite plant parasites: Penicillium and Aspergillus (Aspergillaceae); Plectodiscella (Plectodiscellaceae); Cleistothecopsis, Meliola (Perisporiaceae); Sphaerotheca, Podosphaera, Erysiphe, Trichocladia, Microsphaera, Uncinula, Oidiopsis and Phyllactinia (Erysiphaceae or powdery mildews); and Diplocarpon (Microtheriaceae).

7. Hypocreales with soft leathery, fleshy or membranous perithecia which are *never black* but show various other colors, such as buff, yellow, brown, red or even purple. The perithecia may be free on the substratum, embedded in a mycelial weft or seated on, or sunken in, a stroma. The following are important genera: Nectria, Calonectria, Giberella, Claviceps, Ustilaginoidea, Epichloe and Cordyceps.

8. Dothidiales with the mycelium forming an internal, sclerotia-like, superficially black stroma, becoming exposed by rupture of overlying host tissue and forming perithecia as ascus-containing locules in the stroma, but without differentiated walls. This order includes the black-knot fungi (Dibotryon spp.) and also the genera Phyllachora and Dothidella.

9. Sphaeriales, or the sphere fungi proper, characterized by the formation of typically ostiolate, globular, carbonous perithecia, which may be single or grouped; immersed, erumpent or superficial on the substratum; located on a mycelial weft or on or in a well-developed stroma. The order contains numerous families and genera which furnish many important pathogens in addition to a multitude of economically unimportant, saprophytic species. Some of the more important genera furnishing pathogens are Botryosphaeria, Diaporthe, Endothia, Glomerella, Gnomonia, Guignardia, Mycosphaerella, Numullaria, Ophiobolus, Physalospora, Pyrenophora, Venturia and Xylaria.

PEACH LEAF CURL
Taphrina deformans (Fcl.) Tul.

Peach leaf curl received its name on account of its characteristic effect, but it is known less frequently as "curl," "curly leaf" or "leaf blister."

Leaf curl was well established in European countries by 1880 and may have been introduced from China, the original home of the peach. It has become practically world-wide and is known in all countries where peaches are grown, although it has not developed in certain peach-growing areas in which climatic conditions are unfavorable, for example, the important irrigated districts of central Washington.

Symptoms and Effects.—The leaf curl affects leaves, tender growing shoots and, more rarely, blossoms and fruits but is most conspicuous from its leaf attacks. The diseased leaves are either noticeably reddened or paler in color than normal and become much curled, puckered or distorted, at the same time being greatly increased in thickness, and of a firm or cartilaginous consistency. Only a few leaves of a tree may be affected or the infections may be so numerous as to involve almost the entire foliage, and the individual lesions may include a small portion of a leaf or its entire surface. As the season progresses, the red coloration becomes less pronounced, chlorophyll disappears almost entirely and a grayish bloom or powdery coating (the spores) appears over the upper surface of the affected portions of the leaves. After this stage, the leaves gradually turn brown, wither and fall from the tree in the latter part of June or early in July. Following the loss of leaves, a new set will be produced from dormant buds.

The young terminal twigs may sometimes be involved, being reduced in length, more or less swollen, pale green or yellow and generally produce nothing but curled leaves. In severe infections the blossoms and young fruits are affected, but these are blighted and fall from the tree very early. Fruit infections have been noted recently in Germany (Klee, 1940).

The injury from leaf curl may be summarized as follows: (1) the loss of the foliage in the spring; (2) the death of trees from repeated loss of

foliage through a period of years; (3) the setting of fruit buds for the next year may be reduced or entirely prevented; (4) affected trees are more likely to winterkill; (5) blossoms fail to set fruits or young fruits drop because of direct attacks; and (6) terminal twigs may be killed back or buds are killed in nursery stock. The injury suffered will depend upon the extent and severity of the disease and the general vigor of the trees previous to and following an attack.

Losses.—An estimate of actual financial loss for the country as a whole is rather difficult. At a time when control practices were not well

FIG. 54.—Peach leaves affected with leaf curl. (*Photograph by B. F. Dana.*)

developed, it was estimated that the annual loss to the country would amount to $3,000,000 or more. With the adoption of effective spraying, the losses have been greatly reduced in recent years, but there are still many growers who are either ignorant of the control practices or are willing to chance that the disease will not be serious. It is now over forty years since effective control practices have been known, but the annual loss has recently been estimated by the Plant Disease Survey to amount to as high as 15 per cent in some sections.

Etiology.—Leaf curl is caused by an obligate parasite, *Taphrina deformans* (Fcl.) Tul., a primitive sac fungus, or ascomycete. An intercellular, septate mycelium is found to be quite generally present in hypertrophied leaves and in the cortex of swollen twigs. Three types may be

recognized: (1) *vegetative hyphae*, composed of cells of very irregular form and size, much curved and twisted and of varying diameter, in the parenchyma tissue of affected plants; (2) *distributive hyphae*, or elongated cells, of rather uniform diameter, arranged parallel to the stem axis and found close beneath the epidermal cells of diseased twigs and very abundant in the pith; and (3) *fruiting hyphae*, between the cuticle and the epidermal walls of the upper epidermis. Vigorous, well-developed vegetative hyphae fill the intercellular spaces just below the upper epidermis of

Fig. 55.—Section of a peach leaf affected with leaf curl. Asci containing ascospores are forming on the upper surface; *a*, cuticle of leaf; *b*, bits of mycelium of the fungus; *c*, young asci with spores not yet formed; *d*, spores just formed; *e*, spores being divided into smaller ones; *f*, spores discharging; *g*, empty ascus. (*After Swingle, Mont. Agr. Exp. Sta. Circ.* **37**, 1914.)

the leaf, and branches from these penetrate between the epidermal cells and form an extensive layer of short, densely granular, more rounded cells, the *ascogenous* cells, between the cuticle and the upper epidermal wall. The asci are formed by upward elongations from the ascogenous cells, and a cross wall is formed in each, leaving a stalk or foot portion and the terminal ascus. The subcutaneous and intercellular hyphae are binucleate, but become uninucleate just prior to ascus formation. This upward growth of the developing asci raises the cuticle, and this is either pierced or torn and disappears, leaving the asci exposed to the surface as a more or less continuous plushlike coating, which in section will appear as a palisade-like layer.

The asci are usually flattened or somewhat truncate at the free end, broader above than below, and vary from 25 to 44 μ long by 8 to 12 μ in diameter. Each ascus normally forms eight spores, but the number

may vary from two to eight owing to the failure of some of the nuclei to organize spores. The ascospores are oval to spherical, 6 to 9 μ by 5 to 7 μ, hyaline and surrounded by a firm but rather inconspicuous wall. They are forcibly expelled through an apical slit or rupture in the ascus and may accumulate on the surface of the leaf, giving a white or grayish powdery condition. The ascospores may form buds, which may separate as primary conidia either within the asci or after they have been set free. These primary conidia may continue the budding process and develop secondary conidia. In this way the fungus will produce on solid media a slow-growing, yeastlike, delicately pink colony consisting of budding conidia, short mycelia and resting cells. Ascospores may also germinate direct by the formation of stocky germ tubes. The exact part played by these two types of germination in the production of new infections is somewhat uncertain. The ascospores or the conidia produced from them are borne away from the diseased leaves before they wither and fall.

The ascospores, or the conidia to which they give rise, live through the summer and winter lodged upon the scales of dormant buds and on the bark of twigs and branches. They may also persist in the soil or in the soil cover. In these locations the conidia may continue to increase in numbers by budding when temperature and moisture conditions are favorable. They are resistant to desiccation and have been known to live for more than a year under extremely dry conditions. By this behavior there is present a supply of conidia in the spring of the year to give rise to the new infections. It is the belief that, if conditions at the time buds are unfolding in the spring are unfavorable for infection, the sprout conidia may continue their saprophytic mode of life and still be able to cause infection the next season. This would explain the development of the disease in severe form in a given orchard following a year when it was absent or present only in traces.

Pathological Anatomy.—The first effect of the leaf-curl mycelium is to irritate the host cells and stimulate them to an abnormal activity. In the infected leaf, the cells are increased in size and number and marked changes in form and structure occur. This increase in size and number of cells is most marked in the palisade parenchyma, and the affected cells suffer almost a complete loss of chlorophyll. The increase of cells on either side of the midrib causes a pronounced gathering or cross wrinkling, with the midrib acting like a puckering string. At the same time the affected leaves generally become more or less concave below and convex above, since the palisade parenchyma suffers more hyperplasia than the spongy parenchyma. The mycelium is entirely intercellular, and produces no haustoria, or sucking organs, but does come into very close physical relation to the host cells.

In affected shoots, the cells of the cortical parenchyma are greatly increased in number and size, are more angular than normal and show

variation in the thickness of the walls. The cortex may be eight to ten times as thick as normal. Such affected shoots generally show greatly shortened internodes, with the enlarged leaves crowded to give a plumed, tufted or rosette appearance.

Predisposing Factors.—It has been generally observed that curl is favored by cold, wet weather when the leaves are opening from the bud,

Fig. 56.—Cross section along the line of diseased and healthy tissue in a peach leaf affected with leaf curl. (*After Wallace and Whetzel, N. Y. (Cornell) Agr. Exp. Sta. Bul.* **276**.)

and these conditions are believed to favor the disease for the following reasons: (1) transpiration is checked out of all proportion to root absorption, and the leaf tissue becomes gorged or distended with water; (2) the growth of the host is retarded, while at the same time the moisture and temperature conditions are favorable for the germination and growth of the fungus. In some regions the absence of leaf curl is explained largely by the failure to have proper conditions of either moisture or temperature at the normal infection period. Temperatures of 50 to 60°F. are favorable for infection, but at 70° or above, if leaves are growing rapidly, the fungus may die out even though infection has started.

The general absence of the disease from certain regions like the peach-growing sections of Texas and the Southwest may be explained by the killing of the conidia by the devitalizing temperatures which prevail there during the summer months. This conclusion is suggested by the fact that conidial cultures are "completely devitalized when kept for a few days at 30°C."

The above consideration will perhaps make it clear why leaf curl is more likely to be severe in the neighborhood of large bodies of water, for example, near lake shores or along river valleys. In such localities

there is a greater humidity of the air and the temperature factor in the early spring is more likely to be favorable. Rainfall alone is a minor factor, as may be noted in the Pacific Northwest, where the disease is severe in the region of heavy rainfall west of the Cascades and also severe in certain localities east of the Cascades in which the rainfall is light. Regions of heavy dews, but with light rainfall and early warm spring weather, are unfavorable to the disease.

Host Relationships.—Leaf curl is distinctively a disease of the peach or its derivatives, such as the nectarine and the peach almond. Its occurrence on the almond may be considered very exceptional. Among varieties of peaches great variations are shown in susceptibility to the disease. It seems to be quite generally conceded that seedlings are more susceptible than budded trees, although many budded trees show very high susceptibility.

Pierce made a very exhaustive study of nearly 200 varieties and came to the conclusion that varieties which are reported resistant in one environment may be susceptible when grown under other conditions, and this is substantiated by reports from other countries. Some of the best commercial varieties seem to be very susceptible, for example, the Elberta, a general favorite, and the Lovell, a favorite in California. First consideration should be given to the selection of varieties suitable to their environment or to the trade and only secondary consideration to their behavior with reference to leaf curl since the disease can be effectively controlled.

Control.—It has been demonstrated that in many cases the disease can be effectively controlled (reduced to traces or up to 2 to 5 per cent) by a single application of fungicide—Bordeaux, copper sulphate or lime sulphur—previous to the swelling of the buds in the spring.

Some orchardists and some experimenters have reported that spraying does not always control the disease. These failures may be due to: (1) too late application of the fungicide; (2) lack of thoroughness of application; (3) an improper fungicide; or (4) weather conditions at the time of spraying or immediately following. Good results have been obtained with copper sulphate, 2 pounds to 50 gallons of water; basic copper sulphate, 3–50; different strengths of Bordeaux, as weak as 2–4–100, but most workers recommend the 3–3–50 to 6–6–50; and commercial lime-sulphur (Baumé test, 32°) (1–9 to 1–15). Promising results have recently been obtained by the use of soluble sulphur dusts and by wettable sulphurs at the rate of 16 pounds per 100 gallons.

To be most successful, the fungicide selected must be applied before the buds begin to swell in the spring, as the fungus must be killed before any opportunities are afforded for infection. It was formerly thought that the successful treatments would be obtained only when spraying was carried out during one to three weeks previous to the opening of the

buds, but tests carried out in New York have given successful control when the trees were sprayed in either late fall or winter. Complete covering of the buds is also essential to success; hence best results will be obtained with high pressure in dry, calm weather. While best results will be obtained with spraying before the buds begin to swell, it has been pointed out that delayed spraying after the buds have expanded may be of value if the early spray has been omitted (Groves, 1938).

Since neither plain copper sulphate nor Bordeaux are of any value in the control of San José scale it will be advisable to use lime-sulphur (1–8) as a dormant spray if these insects are present.

The final recommendations for control are as follows: (1) spray *once*, either in the late fall, early winter or preferably in the spring *before the buds start to expand*, using the fungicide that is best suited to the orchard conditions which prevail; (2) spray thoroughly, that is, cover every bud with the fungicide; (3) trees defoliated by leaf curl should be given the best of care and culture in order that they may overcome the drain occasioned by the attack.

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BROWN ROT
Monilinia spp.

Brown rot is a serious disease of stone fruits and, especially in America, is of minor consequence on pome fruits, frequently involving attacks on blossoms, leaves and twigs, larger branches and fruit. One or more of the phases may be present during a single season. The fruit phase has been called mold, gray rot, soft rot, ripe rot and Monilia rot.

Some of the forms of brown rot are known in all of the continents and Japan and New Zealand, in nearly all of the regions where stone fruits are grown, but their occurrence is limited by climatic factors. The disease has been especially severe in the Atlantic Coast states from New Jersey southward; it occurs in the more northern states and southern Canada east of the Rockies but is rare or absent in the more arid Southwest. Certain forms occur in portions of California, and in Oregon, Washington and British Columbia, west of the Cascades, but they are rare or unknown in the dry sections of the Northwest, east of the Cascades, and have never occurred in the important irrigated districts of central Washington.

Since the first careful study of one of the European forms by Bonorden in 1851, the brown-rot fungi have been the subject of detailed study by numerous investigators in Europe, America, Australia and New Zealand.

Symptoms and Effects.—In accordance with the host parts directly attacked, the following phases may be recognized:

1. *Blossom Blight.*—The flower parts turn brown prematurely and, during moist weather, appear soft and rotting and soon show whitish or tawny fungous tufts over the surface of the affected parts, but, under dry conditions following the infection they appear only withered and

Fig. 57.—Blossoms of peach blighted by the brown-rot fungus.　(*Photograph by E. E. Honey.*)

discolored. As the disease progresses the discoloration may extend through all the flower parts and extend down into the pedicels. The blossom blight will vary from traces to a complete blighting of all the blossoms.

2. *Leaf and Twig Blight* (Wither Tip of English Writers).—Direct attacks on young leaves or on succulent shoots may occur just following the blossoming period, causing the affected parts to turn brown. The discoloration spreads from the point of infection until entire leaves or extended areas of leaves and twigs are involved. Affected leaves and shoots shrivel and remain hanging thus presenting a picture similar to frost injury.

3. *Cankers.*—Infections may spread from blighted fruit spurs or twigs into the larger limbs and cause more or less localized dead areas of bark, which later become open wounds. The cracking and splitting of the bark may be accompanied by more or less exudation of gum. The canker may be checked by callus formation, or it may continue to extend until the branch is girdled. Brown rot cankers are common in Europe on cherries, plum and apples, while in America they are common on the peach in the eastern states and less frequent on prunes and pears in the Pacific Northwest.

4. *Fruit Rot.*—Young fruits may be attacked at any period following blossoming, but they are not generally affected until they are approaching maturity. Under dry conditions, but little fruit rot will be evident in the orchard, but rotting may occur in transit to market, while under humid conditions the decay may develop with some rapidity on the ripening fruit still hanging on the tree. Young lesions show a smooth unbroken epidermis but when the lesion has extended, whitish, gray or pale brown sporulating tufts may burst through the skin. The rot advances until the entire fruit is affected, after which it is gradually transformed into a dry shriveled mass, or "mummy," which either remains hanging or falls to the ground. These mummies in the stone fruits are frequently cemented into clusters which remain in the trees during the

FIG. 58.—Peach mummy and branch, showing a young canker caused by the growth of the fungus down the fruit pedicel. (*Photograph by E. E. Honey.*)

fall and winter. Two deviations from this behavior have been described for apples: (1) a *core rot*, with little or no external evidence of the infection (England); and (2) a *black rot*, in which the fruit is transformed into a coal black, smooth shiny, slightly wrinkled mummy with no external evidence of the pathogen. This last condition is produced mainly by late infections.

Losses from Brown Rot.—The injury from brown rot is due to the following: (1) the blighting of blossoms, with consequent failure of affected

trees to set fruit; (2) the blighting of leaves and twigs, with the loss of these as a part of the normal tree; (3) the formation of the cankers which

FIG. 59.—Pears rotted by the common American brown-rot fungus, with concentric rings of conidial tufts. (*Photograph by E. E. Honey.*)

may interfere with the life of a branch or girdle it and cause the death of all distal parts; and (4) the rotting of the fruit in the orchard or after

FIG. 60.—Normal apple and coal-black mummy produced by brown-rot fungus. This is a storage form of brown rot.

harvest. Under extreme conditions, blossom blight may cause almost a complete failure, while a loss of one-third to two-thirds of the blossoms

has been frequently reported for stone fruits. Unsprayed orchards of stone fruits are sometimes almost a total loss due to the rotting of the fruit in the field, while shipments which appear sound at packing time may reach the market in a worthless condition. The extent of the possible losses from the brown rot of peach are emphasized by the estimate that, for the area east of the Rockies, the annual shrinkage in yield amounts to 25 to 35 per cent of the crop representing a valuation of $3,-000,000 to $4,000,000. These losses fall on growers, transportation companies, commission merchants and consumers.

Etiology.—Brown rot is caused by species of Monilinia (Sclerotinia) which produce their conidial or Monilia stages on the various parts of the susceptible hosts, and an apothecial stage on the overwintered mummies (pseudosclerotia). The new generic name, Monilinia, has been proposed to include the species of Sclerotinia which form monilioid conidia and pseudosclerotia. The following are the important fruit-rotting species:

Monilinia fructigena (Pers.) Honey, attacking apples, pears, plums and cherries in England and on the continent, but not known in America.

Monilinia laxa (Aderh. and Ruhl.), Syn. *Sclerotinia cinerea* (Bon.) Schr., on apples, pears, plums, peaches, cherries and apricots in Europe, Asia, Japan and Pacific Coast of North America; forma *mali* represents the variety which produces the blossom wilt and canker of apples in Europe; forma *pruni* and forma *laxa* formerly considered distinct are now merged with the species proper.

Monilinia fructicola (Wint.) Honey, Syn. *Sclerotinia americana* (Worm.) N. and E. and *S. fructicola* (Wint.) Rehm, produces the characteristic effects on stone and pome fruits throughout America and in Australia and New Zealand.

Conidial Stage.—The tufts of the fungus, ashen gray when young and becoming darker with age, appear on the diseased parts under moist conditions and are made up of groups of conidiophores, sometimes rather loosely aggregated but more generally formed into fairly compact pustules. They are not formed until the substratum is well permeated by the mycelium, and then may be sparse or lacking if the air is very dry. The conidia are produced in loose tufts in the common American species but form more definite mycelial cushions in the other species and are developed in articulate branched chains without disjunctors or separating structures as in some of the other species of the genus. Conidia are so variable for different hosts and different growing conditions that the species can rarely be differentiated by spore size (5.7 to 19 by 7.6 to 28 μ represent minimum and maximum for numerous collections).

Microconidia, globose in shape and ranging in size from 2 to 3.25 μ, are produced in chains from flask-shaped conidiophores, formed singly,

in whorls, or in fascicles in compact masses. The microconidiophores may be formed direct from vegetative hyphae, on germ tubes from conidia or ascospores or even directly on conidia or ascospores, both in nature and in cultures, sometimes in copious numbers in the latter.

Apothecial Stage.—The overwintered mummies which have fallen to the ground and may be on the surface or slightly buried in the soil or covered by litter give rise to 1 to 20, or even more, brown, cuplike disks or apothecia 2 to 15 millimeters in diameter, each raised on a cylindrical stalk or stipe. The stipe varies in average length from 0.5 to 3 centimeters (maximum 2 to 3 inches) depending on the depth of soil cover. The campanulate, cup-shaped, flat or irregularly torn and recurved disk bears on its upper face a layer (the hymenium) of erect, cylindrical-clavate, eight-spored asci, mingled with hyaline, septate, simple or branched paraphyses. The hyaline, ellipsoid or ovoid ascospores, uniseriately or subbiseriately arranged in the upper half of the ascus, vary from 5.6 to 16.7 μ long by 2.9 to 8.2 μ wide according to measurements for the common American species recorded by 13 different workers. The ascospores of the European species show but slight differences in form and size.

It was at first believed that apothecia were not produced by mummies until they had passed the second winter in or on the ground, but more recent studies have shown that they may be formed by one-year-old mummies. It is believed that cold is an important factor in inducing apothecial production, as they have been developed in twenty-five weeks from chilled mummies. It has been shown that under certain conditions buried mummies may remain dormant for two to ten years, and produce apothecia when brought to the surface, while under other conditions they may disintegrate in less than ten months. The formation of the apothecia is induced by sufficient moisture, favorable temperatures and proper aeration of the soil.

When the apothecia reach maturity the ascospores are forcibly expelled by the simultaneous explosion of a considerable number of asci, giving rise to "spore puffing." This can be observed by visible clouds of spores which rise from mature apothecia when exposed to drying or drafts of air after being held for a time in a moist chamber. The maturing of the ascospores and their expulsion in nature occurs at a time when blossoms and young twigs of susceptible hosts are in condition to be readily infected.

The source of the spores which produce the first spring infections may be (1) conidial tufts: (*a*) from fallen or hanging mummies; (*b*) from cankers or blighted twigs; and (2) apothecia from the mummies of the last or previous seasons. The first spring infections may be blossom blight or wilt or leaf and twig blight. When the fruit is half grown or

over, the following are the possible sources of the conidia which may start the fruit rot: (1) mummied fruits of the previous season; (2) blossoms blighted in the spring of the current year; (3) blighted leaves killed earlier in the season; (4) blighted twigs killed during the previous season; (5) cankers formed previous to the current season. The statements hold for the three species under consideration except that the conidia of *Monilinia fructigena* do not survive the winter. But few ascospores are viable five weeks after their liberation.

From one or several of the sources conidia are carried by wind, rain and insects to the developing fruits, germinate at once and penetrate

Fig. 61.—Apothecia of the American brown rot developed in the spring from peach mummies. (*Photograph by E. E. Honey.*)

the uninjured epidermis, or through hair sockets, stomata and lenticels, although entrance is facilitated by other fungous lesions (scab) and insect injuries (especially curculio). The first conidial tufts soon appear, and spores are formed, which will be carried to other fruits to continue the work of destruction. The continuation of decay in fruits after picking may be due to incipient infections or to new infections from surface-borne spores.

In a study of 30 strains of *Monilinia fructicola*, Ezekiel distinguished six varieties on the basis of cultural and spore characters, while Seal distinguished at least two physiological forms, but it seems doubtful from the work of Roberts if these represent biological strains.

Predisposing Factors.—Rainy periods with relatively low temperatures at the time of blooming are conducive to blossom blight. Moisture

not only favors the growth of the fungus and the production and germination of spores, but it induces a tenderer and more succulent growth, which is easily penetrated. This applies especially to the fruit phase of the disease, which is rare in a dry season, but severe when cloudy days and frequent rains with hot weather occur as the fruit is approaching maturity.

Transportation losses are affected very much by the temperatures to which the fruit is exposed and, therefore, by the length of time which elapses between packing and refrigeration. The brown-rot fungus makes a relatively slow growth at temperatures of 10°C., or lower, but a rapid growth at higher temperatures until the optimum is reached.

Although uninjured fruits may be entered, infections are facilitated · by breaks and bruises. In many regions where brown rot is severe, peach scab is common and curculio abundant, and these parasites make openings which are common avenues of entrance. Other chewing or sucking insects feeding on susceptible structures may make wounds and even introduce the spores, although these would be washed in by rains if the brown rot is generally prevalent. The curculio has been shown to be responsible for 93 per cent of the infections in some unsprayed orchards.

Host Relations.—Under favorable conditions brown-rot species are probably able to infect all drupaceous and pomaceous species and many other species of the Rosaceae. Peaches, plums, prunes, sour and sweet cherries, apricots, almonds and nectarines are the most important drupaceous hosts, while apple, pear and quinces are all susceptible. The disease is also recorded for the medlar, flowering quinces, flowering almonds, flowering plums, various wild species of Prunus, the rose, grapes, blackcaps and blackberries. The disease is more serious on stone fruits than on pome fruits, but *Monilinia fructigena* is a serious apple rot in England.

Our knowledge concerning the susceptibility of varieties is still rather meager. Summer varieties of apples are reported as very susceptible. Sweet cherries are generally reported as more susceptible than sour cherries. It has been reported that peach varieties covered with a dense growth of hairs are especially susceptible. Resistance in plums has been correlated with a thick skin (epidermis and hypoderm), stomatal abundance, and firmness after ripening. Variable resistance has been noted in apricots with Moorpark and Peach the most resistant (Hesse, 1939).

Susceptibility to brown rot is known to increase with maturity of the fruit. Softening during ripening is due to the solution of the middle lamellae, and "the absence of the middle lamella in fruits which have softened owing to ripening explains the greatly increased spread of the disease at ripening time," since the hyphae advance entirely in the inter- cellular spaces. The idea has been prevalent that young fruits are more resistant because of greater acidity, but it has been shown that the acid content of rotted peaches is greater than that of normal ones.

Control.—The brown rot of pome fruits in America is rarely of sufficient severity to justify special contrôl measures but in many environments the stone fruits must be protected to ensure a crop. No single practice is adequate, but the following should be given consideration:

1. *Sanitary Measures.*—Remove and destroy all blighted twigs; cut out cankered branches or treat the cankers if on large limbs by the removal of all the diseased wood and bark, followed by coating the wound with coal or gas tar; remove short fruit spurs from large limbs; prune trees with a dense head by thinning out, rather than cutting back; thin fruit clusters; remove and destroy affected fruit during the ripening period, and also fallen affected fruit; rake up and destroy rotted fruit after harvest has been completed. The fallen fruit should be buried with a sprinkling of quicklime at least 24 inches below the surface, if curculio is present, otherwise a few inches deep, but without lime is satisfactory. In addition to the above operations in the orchard, the removal and destruction of plum and wild-peach seedling thickets in the vicinity of commercial orchards is recommended.

Commercial pulverized and oiled calcium cyanamid applied to the surface of the soil and vegetative cover with a duster under prune trees at the rate of 220 pounds per acre just previous to the emergence of the apothecia prevented the development of these spore fruits. No injury resulted to apricots, peaches or prunes (Huber and Baur, 1939; Baur and Huber, 1941).

2. *Cultural Practices.*—When plowing and cultivating are consistent with the horticultural demands of an environment, they will undoubtedly be of value. Mummies buried by plowing are not likely to produce apothecia, and those that are near the surface may be hindered in their development by harrowing in the early spring and during the blossoming season.

3. *Spraying or Dusting.*—In regions of severe infestation the use of fungicides must be a regular practice, with variations in the formula and the number of applications to suit local conditions. The following applications have been recommended: (a) the *bud spray* before blossom buds burst; (b) the *calyx spray*, when most of the petals have fallen; (c) the *shuck spray*, when calyxes are shedding or soon after; (d) about two weeks after the shuck spray; (e) the *fruit spray*, one month to two or three weeks before ripening, or two treatments, the first one month before ripening and the second (dusting with sulphur) about ten days before harvest. Lead arsenate, 1 pound to 50 gallons is recommended for all applications except (a) or 5 per cent of lead arsenate if the dust formula is used.

When blossom and leaf and twig blight are not of consequence, the spraying may be begun with application (b) and the fungicide used only

in the last two applications. The recommendation for prunes and cherries in the Pacific Northwest omits application (d) and the insecticide, since curculio is not present.

The following liquid fungicides have been recommended: (a) Bordeaux (4–4–50, or sometimes 3–4–50, 2–3–50 or 1½–2½–50); (b) lime-sulphur (1–50); (c) self-boiled lime-sulphur (8–8–50) or some other sulphur fungicide. Dry-mix lime-sulphur is the best, especially for peaches, since it is much easier to prepare than self-boiled lime-sulphur and also can be used without injury to fruit or foliage. For prunes (a), (b) or (c) may be used; for cherries, (a) or (b), but not self-boiled lime-sulphur on sweet cherries because of its dwarfing effect; for peach and Japanese plums use only (c); for apricots (a), the weaker strength, or (b). Under California conditions, self-boiled lime-sulphur, if used after the fruit had set, caused pronounced dwarfing of apricots. Some promising results have been obtained with apricots and almonds (Wilson and Serr, 1938) by spraying with sodium arsenite, monocalcium arsenite, or zinc arsenite either after the appearance of the conidial fruits or before their development.

Dusting has been used mostly for peaches. The most generally recommended formula is 80 per cent dusting sulphur, 5 per cent arsenate of lead and 15 per cent hydrated lime.

4. *Harvesting and Transportation.*—The fruit if protected by a suitable fungicide and carefully handled in packing and transportation may be marketed at a stage of good quality. Fruit should be placed in cold storage as quickly as possible after harvesting. Fumigation has been tried but no satisfactory commercial results have been obtained, because of injurious effects on appearance or flavor.

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ANTHRACNOSE OF CURRANTS

Pseudopeziza ribis Kleb.

This disease is also called the "leaf blight" and "leaf spot," but should not be confused with the leaf spot of currants due to an entirely different fungus, *Mycosphaerella grossulariae*.

Currant anthracnose is widely distributed in Europe, Asia, Australia, New Zealand and many portions of North America from the Atlantic to the Pacific Coast, including every province in Canada, from Alaska, from Mexico and many of the states from Vermont to California except in the South, but is of more local occurrence than many other fruit diseases and rather variable in the severity of its attacks.

Symptoms and Effects.—The disease first appears on the older and lower leaves causing minute, dark-brown, circular or subcircular spots,

FIG. 62.—Anthracnose (*Pseudopeziza ribis*) on currant leaves.

about $\frac{1}{25}$ inch in diameter, and most conspicuous on the upper surface. The lesions may be few in number or so numerous as to coalesce and cause larger and more irregular dead areas. Minute, shiny, translucent, whitish or flesh-colored spore masses may be noted on the surface of the spots, but these may be washed away by rains. When the leaf spots are few in number, the balance of the leaf may remain apparently normal, but when they are numerous there may be a pronounced chlorosis of the intervening leaf tissue. During some seasons, the disease may confine its attacks to the lower foliage, but in other seasons almost complete

defoliation may result. Lesions may occur also on the petioles and upon the one-year-old canes, being evident on the former as conspicuous, black, slightly sunken spots and, on the latter, as light-brown or pale-yellow, rather inconspicuous spots except when numerous.

In severe attacks lesions on the berries are evident as minute circular black spots resembling fly specks and frequently affected berries may split or crack and considerable shedding may result.

The current season injury is nearly negligible in light attacks, but in severe infestations the yield may be reduced by one-half or more while the fruit may be of inferior quality. When the disease is not controlled

Fig. 63.—*Pseudopeziza ribis.* *A*, section of an acervulus; *B*, small portion of acervulus more highly magnified, showing conidiophores and conidia; *C*, vertical section of an apothecium. (*A, after Stewart; B, after Duggar; C, after Klebahn.*)

in certain environments, the injury is progressive from year to year and plants finally succumb.

Etiology.—Anthracnose is caused by *Pseudopeziza ribis* Kleb., a discomycete or cup fungus, which produces its conidial or Gloeosporium stage on the various lesions during the growing season and its ascigerous stage on the old fallen overwintered leaves. The causal fungus was first named *Gloeosporium ribis* and was known by this binomial until Klebahn found the apothecial form on the overwintered leaves that had been affected with the typical anthracnose. The relationship was demonstrated by the production of typical lesions upon the growing parts by inoculations with the ascospores. The apothecial stage has been found on gooseberries also by Blodgett (1936).

In the parasitic stage the mycelium becomes aggregated into a basal stroma in the center of a leaf spot forming an acervulus, and spores are formed which finally rupture the epidermis and accumulate as the characteristic gelatinous masses described under Symptoms and Effects. The conidia are hyaline, strongly curved or falcate, commonly 19 by 7 μ, but vary from 12 to 24 by 5 to 9 μ in strains from different hosts. The conidia are liberated by the dissolving of the gelatinous matrix, in which they are embedded, by rains, which may carry them to adjacent uninfected portions, and cause new infections when moisture and temperature conditions are favorable for spore germination (10 to 28°C.). The characteristic lesions may appear after ten to fourteen days. Rod-shaped microconidia have been produced in cultures, but show variable form and size, 8.9 by 2.3 μ in some cultures, 11.2 by 1.6 μ in others, and even smaller, or 4.8 by 1.4 μ, in one culture. The role of the microconidia is unknown, but it is probably in connection with fertilization for the initiation of the apothecia.

The infected leaves fall to the ground, but the mycelium becomes saprophytic, persists and organizes apothecia which reach maturity in the spring. They appear as minute, fleshy, disk-shaped structures, partially embedded in the leaf tissue, and break through the epidermis of the overwintered leaves to produce numerous club-shaped asci bearing eight hyaline ovoidal spores, mingled with simple or branched, sometimes one-septate paraphyses. In the spring the ascospores are forcibly discharged and are carried by the wind to the young leaves, and thus early infections result. The early lesions soon begin the formation of acervuli, and, from that time on, secondary infections result from the numerous conidia. To summarize, it may be stated that primary spring infections are induced by ascospores, also by conidia formed in the spring, and probably also by overwintered conidia (Blodgett, 1936).

Exactly what conditions operate to produce an epiphytotic seem uncertain. The disease was very severe in New York in 1889 and in 1901, both seasons being characterized as especially wet, but these observations are opposed to the experience in Bavaria, where the disease was very severe during two rather dry seasons. · It would seem that favorable temperature and moisture conditions during the early part of the season are of more influence than abundant rains later in the season. Some workers report that infection is promoted by an excess of nitrogen and a shortage of potash.

Host Relations.—Anthracnose occurs on both wild and cultivated species of currants and to a lesser extent on gooseberries. Strains from the currant are more pathogenic on currants than on gooseberries and vice versa. Susceptibility of hosts is increased by low vigor. Black currants (*Ribes nigrum* and *R. aureum*) are generally more resistant than

the varieties of red and white currants (*R. rubrum*), although these show varying degrees of susceptibility. In New York, Fay's Prolific, White Grape and Victoria are reported as very susceptible, and the Pearl and Downing more resistant than Smith, Industry, Whitesmith and Houghton. In Prussia red and white Versailles and Fay's Fruitful are reported as susceptible while red Dutch and Erstling von Vierlanden exhibited a high degree of resistance.

Prevention or Control.—Some protection may be afforded by destruction of the fallen leaves by burning or by their burial by cultivation but main reliance must be placed on spraying to prevent infections: (1) from the ascospores produced on the fallen, overwintered leaves; and (2) from conidia developed during the growing season. Good control has been reported by the use of Bordeaux (2–4–50; 4–4–50; and 5–5–50); lime-sulphur (1–40 or 1–50); and sulphur-lead dusting (90 parts finely ground sulphur and 10 parts powdered lead arsenate). Lime-sulphur is less effective than Bordeaux, but in case powdery mildew is present best control of both diseases may be obtained by using lime-sulphur for the first two treatments followed by Bordeaux for the later applications. The value of spraying is increased by pruning to avoid too heavy a growth of canes.

The following applications have been recommended: (1) when the leaves are unfolding; (2) after an interval of 10 to 20 days; (3) late sprayings after similar intervals. The maximum number of applications that has been recommended is six, while cases of successful control have been reported with a single early application or even a single spraying after picking the fruit. These varying results are caused by the seasonal and regional factors that either favor or lessen the severity of the disease.

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ALFALFA LEAF SPOT

Pseudopeziza medicaginis (Lib.) Sacc.

Alfalfa is affected by a number of parasitic fungi which cause a spotting and dropping of the foliage, but the trouble under consideration has been so generally referred to as alfalfa leaf spot, that the name has been allowed to stand. It should not be confused with the yellow leaf blotch (*Pyrenopeziza medicaginis*), which is sometimes even more destructive.

Alfalfa leaf spot was first observed in the United States in 1856 and was recorded as common in Germany as early as 1869. It appeared to be general in Europe by 1909. Since the first published work on the trouble in America in 1890, the disease has become general with the cultivation of alfalfa and can now be found in slight or severe form wherever alfalfa is grown. A very comprehensive study of the disease was published by Jones (1919).

Symptoms and Effects.—The spots on the leaves are brown or very dark, and some will show a central disk which is more frequently on the upper surface. This disk may appear as a jelly-like drop or almost as dark as the surrounding portions of the spot, the former appearance being noted under moist and the latter under arid conditions. The spots may vary in number from a few to a leaflet to a very large number

.Fig. 64.—Alfalfa leaves showing spotting due to *Pseudopeziza medicaginis*.

(50 to 100 or more). With moderate infections the average size is 1.5 to 2 millimeters in diameter, but with heavy infections the spots may remain as mere specks. In many infections there may be more or less chlorosis of the intervening tissue, with the green color persisting longest around the spots. The extremes of the yellowing are likely to occur on the most shaded foliage, even though the infections may not be so numerous as on some leaflets with more exposure. Lesions may also appear on the petioles and succulent stems as elliptical, brown or black spots, 1 to 3 millimeters in length. The characteristics that distinguish this leaf spot from spots caused by other parasites are (1) the circular shape and limited size of the spots and (2) the presence of the small raised disk in the center of the spot when it has reached full development.

The injury from the disease in the established fields is due in large part to the early shedding of the lower leaves, which are the first ones to be attacked. In severe attacks many leaflets may have fallen to the ground before the time of cutting, while many others which were less affected will shatter off during the curing and handling of the hay. Sometimes the final product from such fields is little more than a mass of naked stems, while the really valuable portion, the leaflets, is left behind in the field. Exclusive of the loss from shattering, heavily infected plants yield a crop of lower nutritive value. In New York the first cutting in June may be seriously affected, but it is likely to be more severe on the second and third cuttings, while on the irrigated or upland ranches of the West, where the midsummer rainfall is slight, the first cutting generally shows the maximum infection.

Old established plants are probably never killed outright by leaf spot, but young seedlings are sometimes completely ruined. In some cases where slightly acid soil has lowered the vigor of the young plants, the disease has been noted in unusually severe form. It is probable that the average farmer underestimates the amount of damage from the disease. There is, however, a constant toll, small under dry conditions, but larger under more humid conditions. In some localities experimental plots have been so severely attacked that some were completely destroyed, while losses of 50 per cent have been noted in Iowa, where the disease has been listed as the principal cause of the nonsuccess or the partial success of the crop.

Etiology.—Leaf spot is caused by *Pseudopeziza medicaginis* (Lib.) Sacc., a species of cup fungus which appears to be confined very largely to alfalfa or to other species of Medicago. For a time it was the belief that a similar form, *P. trifolii*, on clovers was identical, but Jones (1919) has presented convincing evidence as a result of pure-culture inoculations that the alfalfa and clover species are distinct.

The disk at the center of the leaf spot is the fruiting body, or the apothecium, of the pathogen. Mature apothecia are 0.5 to 1.5 millimeters in diameter, slightly raised, but sessile and usually surrounded by the torn edges of the leaf epidermis. While the apothecia are usually solitary, some overwintered leaves may show several clustered fruits on a single stroma. The asci are 60 to 70 by 10 μ and the paraphyses continuous, unbranched, swollen at the ends and slightly longer than the asci. The ascospores are uniseriate or irregularly biseriate, continuous, hyaline, biguttulate, irregularly oval and 10 to 12 μ long.

Under conditions of suitable temperature and moisture the spores from mature asci are forcibly discharged by the rupture of the ends of the asci and may be thrown for a distance of several millimeters. They stick readily to surfaces with which they come in contact. Some of the

apothecia will expel their spores during the course of the growing season, and these will serve for the immediate spread of the disease, while others which are retarded in their development, or form later in the season, will persist on the old fallen leaves and be ready to expel spores in the spring.

Spores expelled naturally from asci are capable of germinating at once upon a moist surface. If on a leaf the germ tube is able to pass directly through the cuticle into the epidermal cell and after branching grows into adjacent epidermal cells or down into the palisade layer.

Fig. 65.—Vertical section through an apothecium of the alfalfa leaf-spot fungus (*Pseudopeziza medicaginis*). (*After Combs.*)

In the center of the lesion the palisade cells are partially displaced by the stroma of the fruiting body, "while the cells of the spongy parenchyma are either collapsed or filled with the hyphae." No cicatrice is formed in the outer zone of a lesion, which will explain the absence of the shot-hole effect.

The ascospores are quite resistant to desiccation, according to tests by Jones (1919), since they were able to withstand drying on plaster of Paris blocks for a year and were not injured by freezing. It has been suggested, therefore, that the spores may be able to overwinter on the surface of the seed or on fragments of leaves and calyces mingled with the seed, but the evidence is inconclusive that the disease is seed-borne. It is probable that the first infections in a field are generally started from the wind-blown ascospores, but introduction into new distant localities may depend on carriage with the seed in some form. The presence of a functional conidial stage, as reported by European writers, has not been

verified by recent studies.   Jones has shown that they were dealing with fruits of other fungi.

Control.—In considering the control of the disease the following points should be kept in mind: (1) the ascospores are the only kind of spores that are known to function in nature; (2) these are formed on the leaves, and many of the spore-bearing fallen leaves carry the fungus over the winter; (3) the disease does not spread from clover to alfalfa; (4) alfalfa is the principal host in the regions where alfalfa is generally grown; (5) the pathogen is extensively wind-disseminated; (6) seed disinfection does not prevent the disease in new plantings in regions in which alfalfa is already established.

The recommended control is *early cutting*, which accomplishes two things: (1) the fungus is prevented from maturing its apothecia; and (2) a field is harvested before the shedding of leaves has lowered the value of the crop.   Infected fields should be watched and mowed before the foliage has begun to drop to any extent.

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CHERRY LEAF SPOT

Coccomyces hiemalis Higgins

This disease of cherry is characterized by the production of localized dead spots on leaves, fruit and fruit pedicels, the serious aspect being the defoliation that is likely to result.   The varying effects have suggested such common names as leaf spot, shot-hole disease, yellow leaf and yellows, the two latter because of pronounced chlorosis of the foliage. Leaf spot of cherry was first reported from Europe in 1884 and has been common on both sweet and sour cherries.   Since 1891, when the disease was first studied by Pammel in Iowa, special bulletins on life history and control have been issued from many of the Eastern states and by the U. S. Department of Agriculture.   It is common and destructive from Nebraska eastward to the Atlantic and also in eastern Canada, while in the West it is confined very largely to the humid coast sections of California, Oregon and Washington.

Symptoms and Effects.—Leaf spot first appears on the leaves as small, purple or reddish, circular spots, which later enlarge and turn brown. On certain varieties the brown spots may remain surrounded by a zone of reddish brown or in late infections may remain as small purple spots. The spots, ⅛ inch or less in diameter, may be few in number or they may be so numerous as to coalesce and form large irregular dead areas. During humid periods, whitish spore masses may appear in the center of the leaf lesions, being much more numerous on the lower than

Fig. 66.—Cherry leaves showing spotting due to *Coccomyces hiemalis*.

on the upper surface. The dead brown tissue of the leaf lesions may fall out, leaving either circular or irregular, ragged holes, the shot-hole effect being more frequent and severe on the sour cherries. In severe or later stages of the disease there may be a very marked chlorosis of the leaf tissue between the lesions, thus causing the condition called yellows or yellow leaf. The seriously affected leaves may fall prematurely, resulting in defoliation by July 1 or a little later. Trees, especially the English Morello, which have a weakened vitality from nutritive disturbances or from fungus trunk or root rots, suffer the most extreme defoliation.

Infections may occur on fruit and fruit pedicels and rarely upon the young shoots. The presence of the pedicel lesions, combined with the defoliation, causes the fruit to ripen unevenly. Direct attacks of the fruit are of minor consequence, but fruit lesions may occur as small brown spots. The early shedding of leaves lowers the vitality, and in severe attacks fruit may fail to mature. Wood and bud formation are hindered, and repeated severe attacks may cause the death of the trees. Severely

affected trees are much more liable to winter injury than trees that are free from the disease. The disease may be especially severe on young nursery stock and cause heavy losses unless controlled by spraying.

Etiology.—The common leaf spot of cherry is caused by the ascomycetous fungus, *Coccomyces hiemalis* Higgins, which affects sweet cherries and sour cherries as well as *Prunus pennsylvanica* and *P. mahaleb*. *Coccomyces prunophorae* on plums and *C. lutescens* on chokecherries are related species. The common leaf spot fungus was first described from its conidial stage as *Cylindrosporium padi* Karsh. and was generally known by that name until the discovery of the ascus stage by Higgins in 1914. The detailed studies of Higgins have shown by cultures and inoculations that the acervular or Cylindrosporium stage on the living host is but a part of the life cycle of the ascomycete which produces its apothecial stage on the fallen overwintered leaves.

Fig. 67.—Section of an acervulus showing the Cylindrosporium stage of cherry leaf-spot fungus. (*After Stewart, Cornell Circ.* **21**, 1914.)

Some lesions may fall out before conidial fruits can be formed, but those which persist long enough give rise to one or more acervuli, frequently more common on the lower surface. The acervulus consists of a disk-shaped stroma which forms beneath the epidermis and develops the conidiospores on its upper surface. When these conidia have been formed in sufficient numbers the epidermis is lifted up and ruptured, and they are forced out upon the surface as yellowish-white or whitish-

opalescent, sticky masses or sometimes as more elongated tendrils. The conidia are hyaline, elongate, curved or flexuous, 45 to 60 by 2.5 to 4 μ, and continuous or one- to two-septate.

After the cessation of the formation of the typical Cylindrosporium conidia, the stroma begins to develop and gradually swells toward the lower side of the leaf. During the first warm spring days, the hymenial layer soon becomes evident, asci and paraphyses are formed, and in April or May the asci enlarge and rupture the covering in a more or less stellate fashion. The asci are clavate, 70 to 95 by 11 to 14 μ, eight-spored and have a long stout pedicellate base; the hyaline, continuous or one- to two-septate, linear ascospores are fascicled in the large end of the ascus; and the paraphyses are filiform, septate with apex slightly enlarged

A B

FIG. 68.—*Coccomyces hiemalis.* *A*, section of an apothecium; *B*, asci and paraphyses enlarged. (*After Higgins.*)

and often hooked or forked. Soon after the rupture of the apothecia the mature ascospores may begin to be forcibly expelled from pores in the papillate apex of the asci. Following the exhaustion of the asci, Cylindrosporium-like conidia may be developed from the perithecial stromata.

According to Backus (1933) coiled archicarps develop in stromata following the cessation of formation of conidia, and these produce an elongated trichogyne which grows up to the surface and ends among the microconidia, which are believed to function as spermatia.

The ascus fruits are undoubtedly the source of the first spring infections, since some of the ascospores are set free and carried away by air currents before the first leaf lesions appear in the spring. It has been pointed out that they are more active in producing infections than the Cylindrosporium conidia. The conidia produced in the apothecia follow-

ing the liberation of the ascospores are also capable of producing infections, but their importance in the life history is uncertain. Ascospore discharge occurs only after the overwintered leaves have become thoroughly wet and especially when they begin to dry. Under Wisconsin conditions (Keitt et al., 1937) the discharge was very active during early June and diminished later in the month, but the periods of natural discharge began before blossoming and continued for six to seven weeks. The discharge was influenced by the temperature "being rapid at 16° and higher, distinctly less rapid at 12°, and very sparse at 8, 4 and 1°."

The conidia produced from the acervuli have been shown to lose their vitality rather quickly after drying, but fresh conidia germinate readily under proper conditions of temperature and moisture. During humid periods they are produced in abundance and are washed away by rains or are carried by other natural agencies to healthy foliage and thus serve to spread the disease during the growing season. Apparently the conidia do not live over winter, and there is but little evidence that any active lesions persist on the young twigs. The secondary infections from the conidia occur in successive periods dependent upon the moisture, suitable temperatures and the availability of the inoculum. It is believed that these secondary infections account for the extensive and rapid spread of the disease during the growing season and are responsible for most of the serious injury. The relation of temperature to the percentage of germination of the conidia may be noted as: very slow at 12°C. or lower; much increased at 16°; high at 16 to 28°, with the most rapid elongation of the germ tubes at 20 to 28°; and no germination above 32°C.

The dropping out of the circular leaf lesions with the resulting shothole effect is caused by the enlargement beyond the mycelium of a zone of cells which split away from the central cells. The separated central leaf tissue "turns yellow, shrinks rapidly and soon drops out." It is believed that the enlargement of the cells is due to increased osmotic pressure and that the production of the holes is correlated with the amygdalin content of the leaves. According to the theory the amygdalin is broken down by an enzyme and substances are produced which increase the osmotic pressure. This is substantiated by the absence of amygdalin from Prunus avium in which shot-hole formation is rare.

On the basis of extensive cross inoculations it may be concluded that there is little danger of infection of cultivated cherries by Coccomyces species from wild hosts. Conditions in the Inland Empire offer similar evidence, since Coccomyces is very common on the native chokecherry, but both sour and sweet cherries have remained free from infection even when adjacent to infected chokecherries.

Prevention and Control.—On the basis of life history, two control practices are indicated: (1) the elimination of the ascus stage as the source

of the first infection; and (2) the protection of the developing foliage by the application of a fungicide.

1. *Early Clean Cultivation.*—Turning under as many as possible of the fallen leaves, by clean cultivation before the discharge of the ascospores, should reduce infections and may be used as a supplement to control by spraying when consistent with horticultural practice. To be most effective the clean cultivation should precede the time when the blossom buds begin to break open. If clean cultivation is not consistent with horticultural practice, adequate control can be assured by spraying alone.

2. *Spraying or Dusting.*—In regions in which leaf spot is prevalent in serious form, the adoption of a spray program will be necessary to ensure commercial control. The following applications are recommended: (*a*) beginning when about three-fourths of the petals are off, to be completed as rapidly as possible; (*b*) two to three weeks later; and (*c*) just after the fruit has been harvested. In some southern localities a preblossom spray is reported to be as effective as the petal fall spray (Schneiderhan, 1938). In some seasons and in some localities, applications *a* and *b* and clean cultivation give adequate protection. The spray program should be modified to meet regional variations.

The following liquid fungicides have given good control: (*a*) Bordeaux, (3–4–50) (more rarely 2–3–50), the weaker strength for regions of light infestation; (*b*) commercial lime-sulphur (1–30, 1–40, or 1–50); (*c*) a homemade copper phosphate, only after harvest (Daines, 1904); (*d*) basicop with lime (3–8–100 or 3–6–100) or basicop-zinc sulphate-lime (2–1–1½–100), the latter giving adequate control and no injury (Cation and Robinson, 1938). With any spray an effort should be made to cover both upper and under surfaces of the leaves. Lead arsenate powder (1–50) is used for insect control.

Bordeaux should not be used on sweet cherries because of the danger of injury, but lime-sulphur (1–50) is safe and effective. Bordeaux is reported to cause severe foliage injury and serious reduction in the size of the fruit. It is claimed that the addition of ¼ pound of iron sulphate to each 50 gallons of lime-sulphur will increase the adhesiveness and lessen the danger of burning. Good control, but less effective than with Bordeaux, has been reported from the use of several different brands of flotation sulphur.

Dusting with sulphur has been recommended especially for nursery stock but has not consistently given control in orchard trees. The formula is 90 parts dusting sulphur to 10 parts of finely powdered lead arsenate, applied when the shoots are 8 to 12 inches long, followed by applications at intervals of two weeks during the period of shoot elongation. If preferred, one of the liquid fungicides suited to the variety may be used instead of the dusting sulphur.

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POWDERY MILDEW OF APPLE

Podosphaera leucotricha (E. and E.) Salm.

This disease was first noted in the United States as early as 1871 and is now known to a greater or less extent in all of the apple-producing

FIG. 69.—Shoots of apple heavily infested with powdery mildew (*Podosphaera leucotricha*). (*After Fisher.*)

countries of the world. In some sections it has been serious only on nursery stock, but in a few nonirrigated orchards and in some of the irri-

gated areas, notably certain Pacific Coast orchard areas, the disease has been severe in bearing orchards (Washington and California).

Symptoms and Effects.—The disease is first in evidence on the *leaves* as small grayish or white, feltlike patches, which may rapidly enlarge and soon cover the entire leaf with a powdery coating. In early and severe infections the affected leaves may be killed or may be hard and brittle, reduced in size, narrower than normal, and more or less folded longitudinally.

One-year-old twigs may exhibit the same white powdery appearance as the leaves, but by midsummer (July) this begins to disappear and gradually the external whitish growth is transformed into a brown felt-like covering in which numerous, minute, black fruiting bodies are embedded. The infected one-year-old twigs are either stunted or killed back, either completely or sometimes only at the tips.

As a result of the overwintering of the mildew in the dormant blossom buds, the *entire blossom cluster* and its accompanying leaves may be attacked, causing the floral parts to shrivel and blight without fruit formation.

Young fruits may be invaded soon after the blossoming period by the spread of the fungus from adjacent stems or from secondary infection by the germination of

Fig. 70.—Blossoms and young leaves showing severe attack of powdery mildew. (*After Fisher.*)

disseminated spores. The mildew generally disappears from the fruits by midsummer, but a characteristic, netlike russeting or tracery of fine lines on the skin marks its work. Young fruits showing severe infections may be stunted, and cracks may form which allow shriveling.

The disease has both a direct and an indirect effect on production. The different types of injury are as follows: (1) the leaves are stunted, deformed or killed; (2) affected terminals are either stunted or killed back, wholly or in part; (3) the flowers are deformed or blighted so that fruit may fail to set; (4) the blighting of twigs lessens or prevents the formation of blossom buds for the next season; and (5) the fruit which does mature may be disfigured by russeting or cracking.

Etiology.—Apple powdery mildew is caused by an obligate fungous parasite, *Podosphaera leucotricha* (E. and E.) Salm. Another closely related mildew, *P. oxyacanthae* (DC.) DeBy., is sometimes found on the apple in the eastern United States, but it has never been reported as serious.

The pathogen is an external parasite and spreads its delicate, cobweblike filaments or hyphae (mycelium) over the surface of the affected parts—leaves, flowers, young fruits and one-year-old stems. This fungous body absorbs its nourishment from the underlying cells by means of special sucking organs which penetrate the cell cavities. The fungous body produces two kinds of spores or reproductive bodies—conidia, or summer spores, and ascospores, or overwintering spores.

As soon as the hyphae have established themselves on any parts, the prostrate vegetative hyphae give rise to numerous erect branches which form chains of specialized barrel-shaped cells, or conidia. These conidia soon begin to break away at the ends of the chains and, dropping down between the erect hyphae, produce the characteristic powdery appearance of the affected parts, which has suggested the common name of the parasite. Each conidiophore, or conidia-bearing branch, has an unlimited power of spore production, so that enormous numbers of conidia may be produced on a single leaf. The conidia are carried away by the wind or other agents and can germinate at once to produce new fungous bodies if they can find a new host surface on which to develop. Thus they are responsible for the rapid local spread of the mildew during the earlier portions of the growing period. The optimum temperatures for germination of conidia and infection is 19 to 25°C., but the percentage of infection is low as indicated by even the best germination (50 per cent) (Stoll, 1941). It should be noted that resistance of foliage to infection increases rapidly with age.

Fig. 71.—Apple twigs in dormant condition, showing a heavy coating of powdery mildew. The perithecia are present in large numbers in the dark patches. (*After Fisher.*)

By midsummer the mycelium on the diseased twigs has changed from the colorless, or hyaline, condition to brown, and, as the result of a sexual process, special spore fruits, the *perithecia*, appear in the form of minute, dark-brown, globular bodies, barely visible to the naked eye (75 to 96 μ in diameter), embedded in the external feltlike growth. Each spore fruit shows a number (3 to 11, usually 3 to 5) of long, rigid outgrowths, or appendages, from the upper side and some short, flexuous hyphae (append-

ages) from the lower surface (sometimes nearly obsolete). Young apical appendages are hyaline, thin walled and septate but become thick walled with the lumen more or less obliterated with age, and are dark brown in the lower half but paler toward the tip, which is undivided and blunt or, rarely, once or twice dichotomously divided. The essential structure, however, is a single, oblong or subglobose, eight-spored sac or ascus (55 to 70 by 44 to 50 μ) within the perithecium. The hyaline, single-celled ascospores reach maturity in the spring of the year, and during the warm spring rains the perithecia rupture, the spore sacs protrude and then explode with the expulsion of the ascospores; or the sacs may be expelled and then explode. Thus the ascospores are forcibly expelled, and may be carried away by air currents, and fall on young foliage, where new

Fig. 72.—Perithecium of *Podosphaera leucotricha* with ascus being liberated; also a free ascus much enlarged. (*After Fisher, U. S. Dept. Agr. Bul.* **712**.)

infections may be established. In the apple the perithecia appear to be confined very largely to the twigs but may occur on the petioles, midrib and larger veins. On the pear they are rare on the twigs but may sometimes be produced in abundance on the fruits.

Many attempts to germinate the ascospores have been unsuccessful and it has been demonstrated that they are of little importance, and that the fungus overwinters in the form of a dormant mycelium in the leaf and flower buds. In the spring the mildew commences to grow with the swelling and opening of the buds and thus is in evidence as soon as the young leaves and blossoms appear.

The unimportance of the ascospores in the immediate spread of the fungus is emphasized by the rarity of the spore fruits in certain sections, especially England and the eastern United States, and by the fact that mildew is in evidence in some regions before the ascospores are expelled. It seems especially significant, however, that a copious production of ascospores is characteristic of those regions in which mildew is severe.

The short-lived nature of the conidia, or summer spores, would suggest that they are responsible for the immediate local spread of the disease, while the more resistant ascospores may be responsible for a wider dissemination and a rejuvenation of vigor or virulence.

✔ **Host Relations and Varietal Resistance.**—This mildew affects the pear, quince, cherry, plum, hawthorn and Juneberry besides the apple, its most important host. It is sometimes the cause of serious injury in pear orchards and, although rather inconspicuous on the foliage and twigs, causes black or russeted disfigurations on the fruit. D'Anjou and Louise Bonne are recorded as susceptible, Bartlett moderately susceptible and Flemish Beauty and Winter Nelis markedly resistant.

So far as known, no varieties of apples are immune from powdery mildew, but varying degrees of resistance are shown. In the western United States, 10 commonly grown varieties including Jonathan, Stayman and Spitzenberg are listed as susceptible, while 6 are classed as resistant. In England, 10 varieties are classed as medium in susceptibility, 8 as very susceptible, while 2, Norfolk Beauty and Worcester Pearmain, are classed as highly resistant. Studies in Hungary have shown that susceptible varieties have thinner external epidermal walls than those that show resistance. Physiographic factors influence the percentage of infection so that variety response in different areas is variable.

✓ **Control.**—In the early experiments on the control of apple powdery mildew in the eastern United States, both Bordeaux and ammoniacal copper carbonate were recommended. The development of the disease in severe form in the regions west of the Rocky Mountains presented new control problems, peculiar to the different climatic conditions. Three distinct lines of procedure have received emphasis: (1) pruning; (2) cultural or other practices to produce vigorous early growth; and (3) spraying. In the regions of severe infestation no single practice will give adequate control. In well-cared-for orchards, in which it is possible to use lime-sulphur for scab, mildew causes little or no additional concern.

✓ 1. *Pruning.*—In the winter pruning, interlacing branches should be removed and the long, spindling branches cut back and the gray or silvery, terminal mildewed shoots which carry the fungus over the winter should be cut out to reduce the sources of inoculum. Severely mildewed trees should be pruned much more heavily than healthy trees.

✓ 2. *Cultural or Other Practices.*—Careful attention to cultivation, cover crops or irrigation methods suited to the locality will help by keeping the trees in the best possible vigor. According to some European observations, a deficiency of nitrogen and potash promotes resistance, while much nitrogen and little potash favor the disease. The most satisfactory relation is offered by normal quantities of the essential nutrients or with

a slight excess of potash and phosphoric acid. Others have claimed no increased resistance from potash fertilizer.

3. *Spraying.*—The first two control practices are at best only supplements to spraying, which must be practiced in orchards of the regions favorable to mildew. The time for application of the spray mixtures are as follows: (1) just before the blossoms open (pink spray); (2) just as the last petals are falling (calyx spray); (3) about two weeks after the calyx spray; and (4) about four weeks after the calyx spray. In cases of severe infection, it may be necessary to apply later sprays, which may be put on at intervals of about three weeks until the latter part of August.

In regions where there is no danger of burning, lime-sulphur (1–50) or iron sulphide may be used for all applications, but in regions where sulphur burning is likely use ammoniacal copper carbonate (3–5–50 formula), neutral Bordeaux (4–4–50 formula) or Burgundy mixture with lime (4–5–2–50 formula) for later sprays. Iron sulphide was the most satisfactory spray in the Pajaro Valley, California, but caused injury in the hot irrigated areas of Washington. Lime-sulphur and other sulphur fungicides cause sulphur sunscald when the temperature is 90°F. or above, consequently, the copper sprays listed were adopted (central Washington) to overcome this difficulty. The use of a summer oil following lime-sulphur, has caused defoliation and fruit drop.

Various proprietary preparations of sulphur such as atomic sulphur, colloidal sulphur, etc., consisting of very finely divided particles, are also effective in the control of powdery mildew, especially for protective sprays, but lime-sulphur is more effective when it is necessary to fight well-established cases of active mildew. Dusting with sulphur has given satisfactory control of mildew in nursery stock.

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POWDERY MILDEW OF CEREALS AND GRASSES

Erysiphe graminis DC.

This powdery mildew of cereals and various grass species is world-wide in its distribution but is of most importance on the cereals, especially barley and wheat. As contrasted with the smuts and rusts of cereals, powdery mildew of cereals is of minor importance, but it has caused severe

damage in certain areas, notably in California in 1877 and in Sweden in 1885. It is generally more in evidence in cereal nurseries and test plots than in the general fields.

✓Symptoms and Effects.—The powdery mildew fungus is an external parasite and is first in evidence as superficial groups of a flocculent growth on the upper surface of the leaves, beginning on the lower leaves and spreading to the upper if conditions continue to be favorable. The fun-

Fig. 73.—Semidiagrammatic drawings of haustoria of powdery mildews. A, globular haustorium of *Erysiphe communis*; B, branched haustorium of *E. graminis*; C, haustoria of *Uncinula salicis* in epidermal and subepidermal cells; D, intercellular hypha of four cells of *Phyllactinia corylea* with haustorium from the distal cell. (*Adapted from Grant Smith.*)

gus may appear first as isolated colonies or patches, white when young, becoming powdery with the production of the conidia, but changing to gray or tawny with age. In heavy infections the colonies of mildew may be so numerous as to coalesce and make extended areas completely covered by the fungus. The mildew makes its main growth on the upper surface of the leaf blades, more rarely on the under surface, and in severe attacks may be found on stems and glumes. Following the early growth and copious conidia production, the spore powder diminishes; under favorable conditions scattered black dots, the spore fruits, become evident, generally partly immersed in the fungous patches.✓

✓ The localization of the mildew on the foliage in accordance with its abundance interferes with the normal physiologic processes, especially transpiration and photosynthesis, and infected plants may be stunted

with a reduction in size and number of leaves. The severely infected leaves may dry and shrivel and more or less defoliation result. This loss of functional foliage lowers the storage of food reserves and may cause the heads to be light, poorly filled and prematurely matured, thus reducing both grain and straw yields. According to Graf-Marin (1934), mildewed barley plants transpired 67 per cent more water than healthy plants and in certain tests showed 44 per cent reduction in fresh weight and 23 per cent reduction in dry weight. An increased tendency of mildewed plants to lodge has been reported.

Etiology.—Cereal powdery mildew is caused by the obligate fungous parasite, *Erysiphe graminis* DC. This species is represented by distinct varieties and physiologic races, which exhibit only minor structural or morphological variations.

As noted under the description of symptoms the hyphae are external but obtain their food by the development of special sucking organs, or haustoria, within the epidermal cells. These haustoria are in the form of characteristic fingerlike branches from the central body.

The prostrate vegetative hyphae soon form erect branches, or conidiophores, which give rise to numerous summer spores, or conidia. Each conidiophore with developing conidia consists of a swollen basal cell and a chain of cells which develop into mature conidia. The mature conidia are more or less barrel-shaped and in the different strains or races range in size from 25 to 37 μ in length by 8 to 16 μ in diameter. The chains may consist ultimately of 10 to 12 conidia, but the terminal ones fall off and by their accumulation cause the observed powdery condition. The detached conidia may be spread by wind, rain and other agencies and cause new infections, which will soon develop a new crop of conidia.

Later in the season, sometimes about the time the heads are maturing, the perithecia, or ascus fruits, are formed. "Each perithecium arises as the result of a sexual act, consisting in the fusion of part of the contents of two specialized cells. The mature perithecia are about 200 μ in diameter, globose-depressed, black, and partly immersed in the mycelium. Each consists of an outer envelope of brown, pseudoparenchymatous cells, from the exterior of which simple or slightly branched, pale brown hyphae, known as appendages, arise and an inner cluster of 9 to 30 asci. The asci are cylindrical or ovate, 70 to 108 by 25 to 40 μ in diameter and contain 8 (rarely four) elliptical, single-celled, hyaline spores, measuring 20 to 23 by 10 to 13 μ." (Butler, 1918.)

The conidia germinate best at 12°C. but make the best growth of germ tubes and mycelium at 21°C. After being detached from the conidiophores they soon lose their viability. The conidia form one or more germ tubes, and these develop appressoria from which penetration tubes grow through the epidermal wall and expand in the cells to form

the characteristic digitate haustoria. The fungus cannot penetrate old, well-matured leaves in which the cuticle has become thickened, so it is believed that the immunity of old leaves is merely mechanical rather than physiologic. The increased transpiration of infected foliage is caused by the opening of the stomata and also by the evaporation of moisture through the aerial mycelium. The respiration of mildewed wheat plants

Fig. 74.—Conidial stage of powdery mildew on the surface of a peach leaf. *S*, germ tube from a conidium that has germinated. (*After Tulasne.*)

may be increased as much as 250 per cent, primarily from the activity of the host cells.

The ascospores may not be developed in the perithecia until after the leaves have been killed and fallen to the ground but are available for initiating new infections either in the late fall or winter varieties or on spring seedlings. The mildew may be established on fall seedings by the dissemination of conidia, and winter grain may supply an abundance of inoculum for spring varieties, without the necessity for infections from

ascospores. It has been observed that conidia are readily transported from winter barley to summer barley, especially if the winter barley is at a higher elevation, and that a separation of infected fields for a distance of several hundred yards from new plantings is necessary to limit infection. Danish investigators have reported infections from wind-borne conidia for a distance of 1000 meters.

The severity of powdery mildew is influenced especially by the supply of available nitrogen. An interesting case has been noted in wheat fields of the Palouse country where the strip next to the fence may be heavily infected in contrast to light infection or complete absence of the mildew from the main body of the field. The practice of cutting the fence strip for hay results in a higher supply of available nitrogen and induces a type of growth more susceptible to mildew. The susceptibility to mildew is influenced also by certain soil amendments, for example, lithium chloride or manganese sulphate. By application of the former to wheat, mildew was reduced from 100 to 15 in the treated plot, while the use of 264 pounds per acre of the latter gave a ratio of 5 for the control to 1.45 for the treated.

Host Specialization.—*Erysiphe graminis* affects all our important cereals, barley, oats, rye and wheat, and many other species of the grass family. The species includes *E. graminis hordei* on barley represented by at least seven and possibly nine physiologic races, *avenae* on oats, *secalis* on rye, and two races of *tritici* on wheat with other varieties and races on blue grass, couch grass, brome grasses, rye grasses, etc. This specialization has complicated the problem of mildew control but has offered a fertile field for the plant breeders who have been more concerned with genetic factors than with agricultural values. In barley the breeder is concerned with resistance to the two rusts (*Puccinia anomala* and *P. glumarum*) as well as mildew, and four varieties have been found which combine a fair degree of resistance to mildew as well as to the two rusts (Honecker, 1938). Two selections in Norway (Vik, 1937) have shown high resistance and have been used in breeding, giving a promising cross which has been distributed under the name of Fram. In recent tests of wheat some varieties in each of the series of hard red winter, soft red winter, hard red spring, soft red spring and white spring showed resistance. Emmer and Einkorn were listed as resistant, while *Triticum timopheevi* was not infected (Johnston *et al.*, 1937).

Control.—There is no single practice that is feasible that will control powdery mildew, but some relief may be obtained by the following: (1) early plowing to remove volunteer susceptible plants of the summer crop as a protection to the winter crop; (2) avoidance of heavy or thick seeding; (3) the separation of spring plantings from winter plantings by some intermediate, nonsusceptible crop, or by a windbreak (Pape, 1934);

(4) the selection of the most resistant varieties on the basis of regional experiences; and (5) dusting with sulphur. The degree of damage and the value of the crop would not justify the dusting with sulphur except in the case of nursery or breeding plots.

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ERGOT

Claviceps purpurea (Fr.) Tul.

Ergot is a disease of cereals and wild grasses of outstanding importance, because, in addition to its effect on these hosts, the ergots are of medicinal value and may produce disease known as ergotism in man and animals. The name is of French origin being taken from *argot* or *ergot*, meaning "spur."

The earliest experiences with ergot are connected with its relation to epidemics among people and animals, and it was used as a drug long before its true nature was known. The conidial or honeydew stage was recognized as a fungus by Leveille in 1827, but the sclerotia or ergots had been previously named *Sclerotium clavus* by De Candolle in 1815. The complete working out of the life history of the ergot fungus is to be credited to Tulasne (1853), but many of the important data on etiology have been contributed in comparatively recent times (1903–1930).

Ergot has been reported on some of its hosts from all the continents and New Zealand. In the United States, it has been most frequent on cereals in the region from the New England states westward to the Rocky Mountains. It has been of most importance on rye in countries like Germany, Poland, Russia and Siberia, which produce the greater part of the world supply of this cereal. The importance of rye as a

Fig. 75.—Ergot on wild-wheat grass.

cereal crop may be emphasized by the comparative production of wheat and rye in the United States and Russia and other European countries: wheat 3136 and rye 1631 million bushels for 1929.

Symptoms and Effects.—The disease is not generally noticed in the field until the formation of the *ergots* or *sclerotia*, dark or violet-colored, spurlike bodies, generally longer and larger than normal grains, which usually protrude somewhat from the glumes. The sclerotia, varying from one to many in a head, are more or less cylindric, straight or fre-

quently curved, smooth or longitudinally furrowed, hard or horny, and violet or dark colored on the exterior and white within. The size of the sclerotia depends largely upon the size of the normal grain of the affected host and varies from 3 millimeters in length on a grass host, such as blue grass, to 1 to 3 centimeters in rye and some rye grasses.

Previous to the organization of the sclerotia, the affected grains may be noted by the formation of a sweetish secretion, the "honeydew" which oozes out through the glumes and later as the developing sclerotium becomes evident shows as a sticky mass over the surface of this structure.

Fig. 76.—Ergot on wild rye grass.

A shriveled remnant of this early growth may persist for a time at the tip of the ripening sclerotium but finally drops away.

Ergot is localized, not systemic, and attacks single florets, the actual number of ergotized kernels varying from one to several in a head. The effect of the infections may be threefold: (1) the replacement of the normal grains by the sclerotia; (2) the blasting or sterility of adjacent florets due to incipient infections or to exhaustion of food supply; and (3) reduction in size of spikes or heads. A grain crop is lowered in yield and quality, and a hay crop is lowered in quality.

The disease may be present in traces or a high percentage of the heads may be infected. Under conditions favorable for the disease, 20 to 50 per cent of rye heads may be ergotized, and cases of 70 to 90 per cent

have been recorded (Rumania, 1932). Rye and wheat grasses of the plains country have sometimes contained as high as 5 to 6 per cent of ergot by weight. In cereal crops, a high percentage of the ergots fall to the ground before harvest, but a certain remnant will be included with the threshed grain. A larger percentage generally remain in grasses cut for hay.

Ergotism.—Since early times ergot has been known to cause disease in both man and animals. Epidemics were fairly common in the Middle Ages but have become more rare in the last few centuries. Even in recent years, cases have been reported among the peasant classes of Europe, and, in the epidemic of 1926, eleven thousand persons were reported as suffering from the nervous type of poisoning. Two types of ergotism in man are convulsive, or nervous, and gangrenous (for further details, see Barger, 1931). The active principle is an alkaloid. Four different names have been used, ergometrine, ergotocin, ergobasine and ergostetrine, but these compounds appear to be identical (Karsch, M. S., *et al.*, 1936).

Even during recent years the feeding of ergotized grain or hay has caused losses to stockmen as follows: (1) when consumed in quantities too small to give rise to the pronounced symptoms of ergot poisoning, by the impairment of the general health of animals; (2) from the production of abortion in cows and mares; (3) from either spasmodic or gangrenous ergotism, when ergot is present in considerable quantities or is consumed in smaller quantities through a considerable period. In the latter the gangrene may cause a loosening and sloughing off of the hoofs, tips of ears, tip of the tail or a shedding of teeth and hair. This gangrene progresses and the affected parts shrivel, harden and finally drop away without pain. Affected animals gradually become more and more emaciated and death results. Other species than *Claviceps purpurea* may poison livestock. A notable illustration is *C. paspali*.

Etiology.—Ergot of rye and other cereals, as well as a number of wild and cultivated grasses, is due to *Claviceps purpurea* (Fr.) Tul., an ascogenous fungus belonging to the *Hypocreaceae*. This fungus produces three distinct stages in its life cycle: (1) the vegetative mycelium, which permeates and destroys the young ovaries and produces a conidial stage on the surface of the young fungous growth (the *sphacelial stage*); (2) the matured ergots or *sclerotia*, which are dormant or resting structures for carrying the pathogen through the winter period; and (3) the ascigerous stage, which is formed when the overwintered sclerotia germinate.

The sclerotia when mature either fall to the ground or are mingled with the grain and may be returned to the field with the seed. It has been reported that all the large sclerotia and 80 per cent of the medium-sized fall to the ground, while all the small sclerotia remain in the head. Dur-

ing the following spring, the sclerotia which are lying on or in the soil
germinate by the production of one to several stromata, each consisting of
a slender, reddish, pale-violet or whitish stalk or stipe surmounted by a

Fig. 77.—*Claviceps purpurea.* *A*, hypha with conidia; *B*, head of rye with several
mature sclerotia; *C*, a germinated sclerotium with several perithecial stromata; *D*, section
of a perithecial stroma showing the numerous peripheral perithecia; *E*, section of a single
perithecium much enlarged; *F*, ascus with eight filiform spores; *G*, ruptured ascus with
ascospores protruding; *H*, a single ascospore.   (*A, B, after Strasburger; C–H, after Tulasne.*)

reddish, flesh-colored or pale-fawn-colored globular head, the perithecial
receptacle or *sphaeridium*, the surface of which is covered with minute
elevations, the projecting ostioles of the perithecia sunken throughout

the entire periphery. The stipes are from ¼ to 1 inch in length, varying with the depth of the sclerotium below the surface of the soil, and the capitate sphaeridia are double their diameter. The perithecia are flask-shaped cavities, with walls scarcely different from the surrounding fungous tissue of the receptacle, and each opens on a surface papilla by a narrow ostiole. The perithecia are filled with curved hyaline asci, narrow below and above and broader in the middle, surrounded by hyaline, club-shaped periphyses which differ but little from the asci in form. Each ascus contains a fascicle or bundle of eight, slender, hyaline, needle-shaped spores, 50 to 76 μ long.

The ascospores are forcibly ejected from the asci and may then be carried upward by air currents (convection currents) or they may be swept away by the wind. It has been proved that under field conditions, with still, moist air, convection currents could carry the expelled ascospores to the height of rye heads, and so ascospores could reach the flowers without the intervention of rain, insects or wind.

The wind-blown ascospores reach the young ovary of an open flower, germinate and the resulting hyphae soon pervade the tissues of the ovary and destroy them, leaving nothing but a fungous growth which maintains the general shape of the ovary. The surface of this fungous body is channeled or thrown into numerous folds or convolutions covered with short conidiophores, which produce enormous numbers of hyaline, ovate or sphaeroidal conidia, 0.7 to 3.5 μ. During this formation of conidia there is a copious secretion of the "honeydew," noted under Symptoms, and this becomes filled with the conidia, which are detached from the conidiophores and float out upon the surface. This *sphacelial stage* is visited by insects which carry the conidia away to other flowers and thus spread the fungus during the flowering period. As growth progresses, the development of conidia gradually ceases, first in the basal portion. Here the mycelium becomes gradually compacted, the structure thickens and the superficial hyphae turn reddish and then violet colored. This change progresses gradually upwards until the entire sphacelial stage has disappeared.

It has been pointed out that the sclerotia from certain grasses show special adaptations for dissemination. In *Brachypodium sylvaticum* and in *Calamagrostis epigeios*, the ergots are disseminated by the same devices as the host seeds—by barbed awns in the former, and by a parachute or hair tuft in the latter. Grasses which grow normally in wet or marshy places or along the banks of streams, when affected by ergot, produce "swimmers" or sclerotia which will float on water and can endure a long sojourn in water without decaying, while the ergots of strictly terrestrial grasses sink in water at once, and soon decay if kept immersed. The "swimmers" owe their buoyancy to a greater content of air.

Infection, whether primary from ascospores, or secondary and from conidia, can take place only during the flowering period of the host. The actual infection may be before pollination, or even after fertilization has taken place, but only in the young condition of the ovary. The principal seat of infection is not the stigmas or the free wall of the ovary but the point of insertion of the ovary. Spores may germinate on the stigma and produce long germ tubes which grow down, encircling the ovary, and enter at its base. The infection period is then relatively short for any single floret, but is prolonged for the host as a whole by the successive maturing of different florets. It is evident that ascospores, developed from the overwintered sclerotia, are responsible for the first or primary infections. Even though the conidia of the sphacelial stage are known to preserve their infective power for at least a year if kept dry, they are not believed to survive the winter under natural field conditions. Florets infected from ascospores produce honeydew and conidia after a short incubation period, and these may be carried by insects to other flowers and new infections may result as long as there are any flowers in the susceptible stage. A few primary infections with ascospores may, therefore, be sufficient to cause general infection, provided conditions are favorable for the development of the sphacelial stage and the dissemination of the conidia.

Sclerotia are able to germinate the season following their production, but it seems to be uncertain just how long they remain viable. One-year-old sclerotia appear to be unable to germinate unless they have been subjected to low temperatures. Maximum germination is obtained by 30 to 40 days' exposure to close to zero temperatures. The optimum germination temperature is 18 to 22°C.; the minimum, 10°C. It has been shown that two-year-old sclerotia, if they failed to germinate the first year, are still viable, whether kept dry or subjected to conditions favorable for growth. Fragments of sclerotia may germinate, and those which fall in the field will germinate at the same time as those that are planted later. These facts have an important bearing on the introduction of ergot with contaminated seed.

Predisposing Factors.—In certain sections of the world ergot is present every season in small amount but, with favorable conditions, develops in epiphytotic severity. The atmospheric conditions during the flowering period are of prime importance. The especially favorable conditions are reported to be a rainy spring, a dry sunny period just before and at the beginning of the flowering period of the rye, and then rain and lower temperatures which will prolong the flowering period and keep the glumes open in order to receive the pollen from other plants and thereby prolong the period of susceptibility to infection by ergot. It is claimed that the greater susceptibility of rye as compared with other cereals is due to the

fact that rye depends largely on cross-fertilization instead of self-fertilization as in wheat or barley. It has been reported that the greatest amount of ergot develops with (1) a relative humidity at flowering time of 74 per cent or higher; (2) a temperature at flowering of 13 to 15°C.; and (3) a flowering period of fourteen or more days.

It is generally agreed that grasses in low, damp ground are more likely to be ergotized than those growing in dry soil. Ergot is frequently common along stream valleys, on the north border of woods, on north hillsides in protected coves and in shady places, since such localities favor the development of the ascigerous stage and the dissemination of the ascospores. Ergot is frequently more severe in protected valleys which are free from the sweep of strong winds than in the open windswept plains. It has been noted that broadcasted grain is more subject to ergot than drilled fields, and this may be explained by the deeper seeding of uniform depth, which will shorten the flowering period and would probably bury the ergots too deep for the production of the perithecial stage.

Host Relations.—In addition to the various wild and cultivated grasses, *Claviceps purpurea* affects rye, wheat, oats and barley. It is most common on rye and is only rarely of economic importance on the other cereals. Heavy infection of wheat occurred in North Dakota in 1921 and in France in 1922; and it has been prominent as a disease of oats in Algeria (1922). Barley varieties show considerable variation in susceptibility: the variety *erectum* among the two-rowed barleys, which rarely has open flowers, is very resistant; and Hannchen, with numerous open flowers, is especially susceptible.

The following biologic or physiologic races of *Claviceps purpurea* have been recognized: (1) the rye form, which infects in addition the other cereals and various wild and cultivated grasses; (2) a form on *Anthoxanthum odoratum*, which attacks rye and some other grasses but not barley; (3) a form on *Brachypodium silvaticum, Milium effusum* and several other grasses, which does not infect rye; (4) a form on *Lolium perenne*, English rye grass, which infects *Bromus erectus* and other Lolium species but not rye; (5) a form on *Glyceria fluitans*, which is confined to that species. It seems probable that there are other races which have not yet been differentiated. In addition to *C. purpurea* and its races, 20 other species occur mostly on wild grasses and sedges.

Control.—Of the three following control practices the first is of most importance (1) *The removal of the ergots from seed grain.* Screening, sifting or fanning removes some but complete removal is possible only by sedimentation, using a salt solution. The grain is stirred up in a 20 to 32 per cent solution of common salt or 32 to 37 per cent solution of potassium chloride, which permits the ergots to float to the top, when

they may be skimmed off or decanted. The cleaned grain is then washed in water and dried. (2) *Sanitary practices.* Clean out susceptible grasses from the vicinity of cereal fields or mow before blossoming, or cut meadows early. Burning of wild-hay lands may kill some of the matured ergots. (3) *Cultural methods.* Rye should not follow rye if a previous ergotized crop was produced, and a mixed early and late rye or a close planting of early and late rye should be avoided. Deep plowing and drill seeding will bury residual or seed-included sclerotia and thus prevent the development of the ascus stage. (4) *Selection of varieties.* Those with a short regular flowing period will be least infected.

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BLACK KNOT

Dibotryon morbosum (Sch.) T. and S.

Since its first recognition over a hundred years ago, black knot has continued to be a serious disease of plums and cherries in certain portions of the eastern United States and Canada. It is believed to be of American origin, probably affecting wild species from which it spread to cultivated varieties. The disease spread westward, but it never became as severe in the Middle West as on the Atlantic seaboard. The disease is rare west of the Rocky Mountains, except on wild species. The first complete account of black knot was published by Peck in 1872, while a fuller discussion was presented by Farlow in 1876. Other contributions followed in succeeding years, up to 1914, when a period of inactivity followed until the work of Koch (1933–1935).

Symptoms and Effects.—Black knot is first in evidence as a slight swelling of a twig or branch, either adjacent to an old knot or separate from it. Primary or new swellings may be observed in the fall, but they become more conspicuous the next spring after growth starts. The bark on the swellings ruptures and a straw-colored or light yellowish-brown, granular growth fills the crevices. As the season progresses the overgrowth becomes more pronounced and somewhat darker. In the

late spring or early summer, the smooth surface of these excrescences shows a pale-greenish tinge at places, and soon the entire surface becomes an olive green and appears covered with a velvetlike pile. This velvety surface soon disappears, the knot becomes darker, and by late fall it has become perfectly black.

The knots vary in location, shape and size. They may appear on young twigs or on older branches up to about 2 inches in diameter, and

Fig. 78.—Black knot on Japanese plum.

may be located at any of the following places: (1) crotches of limbs; (2) at the union of the growth of consecutive seasons; (3) on fruit spurs; (4) at the tips of twigs; and (5) in the axils of leaves or about an axillary bud. Some of the knots may be short ($\frac{1}{4}$ to $\frac{1}{2}$ inch), but they generally extend for some distance along a branch (several inches to a foot), and are more or less fusiform, although they may sometimes be nearly uniform in diameter for the entire length. Adjacent knots may sometimes fuse to form much more extended excrescences. Their diameter depends largely on the size of the structure on which they are located, so that they

may be only a fraction of an inch or 1 or 2 inches in diameter. It is typical for the knot to be confined to one side of the twig or branch, yet at times the branch is completely encircled. The affected structures may be straight or curved or even thrown into more or less of an irregular spiral.

In a tree in which the disease has been allowed to run its course undisturbed for a number of years, the following should be found if the tree is examined in June: (1) young excrescences developed during the spring growth; (2) old knots that matured during the early spring; and (3) old knots developed during previous seasons, which are likely to be more or less eaten by insects and are frequently infested with saprophytic fungi. Some of the new knots may be extensions of old ones rather than representing new infections.

The infections with black knot may kill small branches in one year, but in other cases the branch may persist and the knots gradually extend from year to year and girdling may result with the death of distal parts. The amount of injury depends upon the number and location of the knots and their age. A Lombard plum tree in which the disease was allowed to run its course for four years contained 1061 knots (Koch). Because of the ravages of black knot the growing of plums and cherries has been abandoned in some localities and made hazardous in others.

Etiology.—The disease is caused by the ascomycetous fungus *Dibotryon morbosum* (Schw.) T. and S. which produces its *conidial* or Hormodendrum stage on the young knots giving them the velvety, olive-green color as described under Symptoms, and an *ascigerous* stage on the surface of the black matured knots. Many different names have been given to the parasite since its first study, but *Plowrightia morbosa* has been used in most American literature, until the present name was proposed. This change is desirable as the black-knot fungus does not fit the original Plowrightia concept. Successful inoculations have been made using either ascospores or conidia and also with host tissue containing mycelium. According to the most recent work, 84 per cent of the knots produced from ascospores and conidia were from inoculations in the month of May, and 26 per cent infection was obtained in that month.

Knots may be *primary*, that is, the direct result of infection from spores, or *secondary*, due to the invasion of new portions by the mycelium that is already present in the tissues from old infections. Primary infections, at least many of them, produce conidia during August and September of the year of infection, and perithecia during the following winter and spring, thus completing the life cycle in one year, instead of two as was formerly believed. The mycelium in a certain per cent of established galls remains active and produces an extension of the gall which develops the conidial and ascus stages in the regular sequence.

The mycelium pervades the tissue of a knot and in the spring finally forms a superficial stromatic layer, or pseudoparenchyma, of closely aggregated fungous cells, which become covered with a dense coating of erect, septate, somewhat branched conidiophores (May and June), 40 to 60 μ long. These produce terminal or lateral, brown, continuous, ovate

Fig. 79.—*Dibotryon morbosum:* a, habit sketch; b, section of stroma and perithecia; c, conidiophores from the surface of a young knot with several conidiophores and spores more highly magnified; d, section of a single perithecium, an ascus and paraphyses, and several ascospores germinating. (*After Longyear.*)

conidia, about 4 to 8 by 2 to 5 μ. Branched conidiophores with the conidia in chains are produced in culture giving a typical Hormodendrum type of growth, with conidia 3 to 20 by 2 to 5.50 μ. A very similar development may be induced on the knot under suitable conditions of moisture. The conidia separate easily from the conidiophores, are scattered by wind and rain and have been found in viable condition on the bark and buds throughout the balance of the season and through the winter. Under certain conditions, thick-walled resting spores or *chlam-*

ydospores are formed when the conidia germinate in intimate association with antagonistic bacteria.

As the season advances, the conidial production gradually ceases; the primordia of the perithecia, developing beneath the conidiophores, gradually become evident as minute hemispherical protuberances; internal organization is proceeding; and finally the perithecia are formed covering the surface stromatic layer. Each perithecium is a single, ostiolate cavity within a stroma-like aggregate of fungous tissue and is filled with asci, 120 μ long, containing eight, hyaline, one-septate, uniseriate or irregularly arranged ascospores, 8 to 10 by 16 to 20 μ, the lower cell being uniformly shorter and narrower than the terminal cell. Sterile, filiform, nonseptate filaments or paraphyses, with slightly enlarged tips, are mingled with the asci. Mature ascospores have been found in the latitude of Ontario as early as February, but they are not discharged until the spring rains.

The ascospores are forcibly expelled after reaching maturity (from March to early June) whenever moisture and temperature conditions are favorable. When the knots are wet by rains, expulsion of ascospores begins at about 36°F., increases with rise in temperature, is heavy at 50 to 80°F. but, above 88°F., is negligible. The ascospores are air- or windborne following periods of expulsion, and may be carried to distant trees.

Artificial infections have been secured only during May and early June, and it seems that natural infections are probably largely restricted to this time. It appears to be essential that the proper temperature and moisture factors be present combined with the condition of host tissues found only during this restricted time. Viable spores and suitable moisture and temperature conditions are afforded at other times, but the period of host receptivity may be past. The idea formerly prevailed that wood of all ages is suceptible to infection, but from recent work (Koch) it may be concluded that nearly all primary infections are on current-season wood.

Under natural conditions, certain imperfect fungi may be found associated with conidia-bearing or mature knots, either as parasites or as saprophytes on the dead tissue. Several of these forms were at first thought to belong to the life cycle of the black-knot fungus. *Cepalothecium roseum*, causing a delicate pink coloration, establishes itself on the conidia-bearing stromata, as an active parasite; a *Coniothyrium* species, a pycnidial form with brown, continuous spores, is a very common accompaniment, beginning on young galls and continuing on the perithecia with indications of parasitism; *Hendersonula morbosa*, an acervular form with yellowish septate spores, is not uncommon in some localities; and less frequently, a number of other imperfect fungi are found on the knots.

Host Relations.—Black knot affects various species of plums and cherries, and the opinion has been expressed that there are no varieties of cultivated plums that are not subject to the disease. In general, cherries are reported to suffer less than plums, the sour cherries much more than sweet cherries, although their behavior seems to vary with the environment. The disease is especially in evidence on many wild species but is sometimes noted on one species when an adjacent species, perhaps even with interlocking limbs, remains free, and wild and cultivated species may be in proximity without both becoming infected. Very contradictory statements as to susceptibility of varieties have been made in different parts of the country, consequently, it would seem unwise to attempt any specific statements as to varietal relations. The probable explanation for this behavior is the existence of biological strains or species which have become so adapted to certain hosts that they are unable to infect others. It seems certain that the black knot of the native chokecherry (*Prunus demissa*) in the Pacific Northwest is a distinct biological form, since all cultivated plums and cherries in the same environments remain free from the disease. Morphologically distinct strains of the conidial stage, differing in spore size, growth rates and growth characters, have been obtained from monoascospore cultures, and some differences have been recorded for ascospore size on different hosts.

Control.—Three general practices for the control of the disease must be adopted: (1) Cut out and destroy thickets of wild cherries or plums adjacent to cultivated orchards, if such are infected, unless the strain does not affect the cultivated species. (2) In the winter or before growth starts in the spring, prune out all knots on cultivated species, cutting off the limbs several inches below the external evidence of the swellings, or cut out the knotted tissue from large limbs, at least ½ inch beyond the boundary of the knot and into the outer xylem. In both (1) and (2) destroy the prunings by burning at least before growth starts in the spring, since, if left, they would produce ascospores capable of causing infection. (3) Spraying. Combined with the surgical treatment the timely application of a fungicide has given more than 95 per cent control. A delayed dormant spray of 3 per cent oil-emulsion Bordeaux (3–6–50) or 1–8 lime-sulphur, followed by 1–40 lime-sulphur when the buds are breaking; and by the same strength when the shucks are falling have given good results. Excellent control has also been obtained by an additional application of lime-sulphur (1–50) during full bloom.

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Venturia inaequalis (Cke.) Wint.

The disease known most frequently as "apple scab" is also referred to as "scurf," "black spot," "black-spot fungus," "Tasmanian black spot," "black-spot scab" and "rust." Black spot is the name most frequently employed in England, Australia and South Africa, while scab is the name most frequently used in America.

FIG. 80.—Apple leaves showing heavy scab infections, diffuse or spreading type on the left, localized spots on the right. (*After Wallace, N. Y. (Cornell) Agr. Exp. Sta. Bul.* **335.**)

Scab has been known to botanists since the first part of the nineteenth century and has spread to all parts of the world where apples are grown. It varies in its severity with the climatic conditions, being favored by abundant rainfall and the proper temperature relations. There are but few important apple-growing regions in the United States in which scab is either absent entirely or present in such slight amounts as to render control measures unnecessary. The most favored of these regions are the famous Yakima and Wenatchee Valleys of central Washington. Since 1914, when one writer listed 505 different publications dealing with

some phase of scab, a voluminous literature has continued to accumulate, the emphasis being placed upon epidemiology and control. This latter phase has offered an apparently fertile field for periodic reports from a high percentage of the plant pathologists in the infested regions, and

FIG. 81.—A normal apple and others variously spotted, deformed or atrophied as a result of a severe attack of scab.

FIG. 82.—Apples with large scab lesions showing deep cracks.

apparently this practice will continue through all time! Over 265 contributions have been recorded during the last ten years.

Symptoms.—Scab attacks leaves, blossoms and fruits, and sometimes appears on the young twigs:

Leaf infections in the early part of the season show as more or less circular, brownish or gray spots, which later may become olive green or nearly black. Young infections frequently show radiating dendritic

ramifications of the fungus, easily visible with the hand lens. Some of the deviations from this symptomology as induced by severity of infection or seasonal conditions are: (1) hypertrophy or thickening of the lesions; (2) smooth or dull surface of old lesions; (3) some irregular perforation of lesions; (4) a uniform brown coloration or a scorched appearance of extended areas, producing a diffuse form; (5) reduction in size of leaves with more or less curling or distortion; (6) numerous lesions with intervening chlorosis resulting in heavy leaf fall.

Fruit lesions first show as small, more or less raised, brown or black, somewhat circular spots beneath the skin, which later break the cuticle, thus exposing the nearly black, velvetlike sporulating fungus. Some of the deviations from this picture on the fruit are: (1) the bare, brown center of older spots; (2) the corky or scablike character of old lesions; (3) malformation, cracking or reduced size of the fruit; (4) the coalescing of adjacent lesions to form extended scabbed areas; (5) pin-point scab, or small black lesions appearing late in the season; (6) "storage scab," or lesions which appear after harvest. In some of the two latter types of lesions the cuticle may not be ruptured.

Blossom lesions may develop on pedicels, calyx, petals or very young

Fig. 83.—One-year-old apple sprouts showing numerous scab lesions.

fruits and, in extreme cases, cause a shedding of the developing fruit.

Twig infections, ew and scattered or so numerous as to coalesce, and similar to those on the fruits, may appear on the bark of one-year-old shoots. When young, these may show sporulation, but, later in the season, the sporulation may cease and the bark show a more scaly character. Twig lesions have been called "grind or scurf" by German writers and are more common in pear scab than in apple scab.

Effects.—Scab lowers both quality and quantity of the commercial product, the fruit, both by current-season injury and by carry-over effects to the next season.

The development of scab on the *foliage* may be responsible for: (1) more or less defoliation if lesions are numerous or if the infection is diffuse; (2) reduced photosynthetic work or food manufacture in proportion to the amount of leaf surface invaded. This foliage attack is devitalizing by reducing the manufacture of carbohydrate food and the influence may even carry over to the following year.

The attacks of scab on the *blossoms and fruit* take the largest toll. In extreme cases infections on blossoms, fruit pedicels or young fruit may cause a complete blighting or dropping of fruit while still very small and, in the average scab year, cause some reduction in set of fruit. In the less

Fig. 84.—Conidial stage of *Venturia inaequalis*. Section from the edge of a fruit lesion, showing uplifted cuticle, fungous stroma, conidiophores and conidia. (*After Wallace, N. Y. (Cornell) Agr. Exp. Sta. Bul.* **335.**)

severe attacks the fruit may persist through the growing season and, according to the degree of infection, may show one to numerous lesions, with or without deformity and reduction of size. Severe development of scab is a prominent factor in producing premature dropping of the fruit. Severely affected fruits may be relegated to the culls, while more moderate infections lower the commercial grade.

Scab affects the keeping quality of the fruit by (1) causing the fruit to shrivel faster than sound fruit owing to water losses amounting in extreme cases to three and one-half times those of normal scab-free fruits; and (2) by permitting the entrance of rot-producing fungi through the scab lesions. Storage scab may make it necessary to re-sort and pack after the fruit has gone into storage.

Etiology.—Scab is due to an ascomycetous fungus, *Venturia inaequalis* (Cke.) Wint., which lives as a parasite (the imperfect or Fusicladium

stage), on the leaves, twigs and fruits of the apple and completes its life cycle as a saprophyte (the perfect, perithecial or Venturia stage) upon the dead and fallen leaves of the same host.   All of the earlier publications dealt only with the Fusicladium stage, and it was not until the work of Aderhold in Germany in 1894 and Clinton in America in 1901 that the two stages were definitely proved to represent the life of a single organism.

FIG. 85.—Section of a perithecium from an overwintered fallen leaf. (*After Wallace. N. Y. (Cornell) Agr. Exp. Sta. Bul.* **335.**)

The mycelium on the leaves is at first subcuticular and grows radially with a characteristic dendritic branching and later forms one or more layers of closely aggregated cells, thus producing a stroma-like growth which breaks the cuticle and permits the production of numerous erect conidiophores exposed to the surface.   In the initiation of an infection the spore germinates to form an appressorium closely adherent to the

cuticle, and this organ forms a very tenuous infection hypha from a minute pore. This hypha pierces the cuticle, and the distal end enlarges to form a primary hypha which continues the development in close contact with the outer walls of the epidermal cells (Nusbaum and Keitt, 1938). The older mycelium and conidiophores are brownish and the numerous spores and conidiophores give the scab spots a powdery or velvety appearance and an olivaceous or almost black color. On the fruit the stromatic cushions of mycelium frequently completely destroy the epidermal cells and extend deeper into the pulp tissue. The conidiophores are short, erect, straight when young but more or less flexuous with age, olivaceous and continuous or one or two septate. The conidia are lanceolate or ovate, with the base generally somewhat truncate, continuous or more rarely one septate, 12 to 22 by 6 to 9 μ and are produced in indefinite numbers from each conidiophore. The cells of the mycelium, the conidiophores and the conidia are all uninucleate.

The mycelium continues its development on the dead fallen leaves but, as a saprophyte, penetrates into the mesophyll and organizes the *perithecia* or ascus fruits which reach the sporulating stage during the spring of the following year. They appear mostly on the under surface of the leaves as spherical or subspherical bodies, 90 to 100 μ in diameter, embedded in the leaf tissue but slightly protruding on the surface. The mature perithecium is ostiolate, provided with a wall of several layers of dark cells and produces an indefinite number of oblong, spatulate, eight-spored asci or spore sacs. The ascospores are two celled, 11 to 15 by 5 to 7 μ, olive brown, with the upper cell wider than the lower one.

Following the warm spring rains, the asci in succession elongate, protrude through the ostiole, explode and thus forcibly expel the ascospores which are then carried away by the wind or air currents. Some may reach the young leaves or blossom buds and produce the first or primary infections. The periods of spore discharge vary with the season and the number of rain periods from a few to as many as 16 in a single season. The primary infections soon give rise to conidia which may be scattered largely by rains to other host parts and give rise to secondary infections, and both primary and secondary infections may furnish conidia to produce new infections throughout the season whenever moisture and temperature conditions are favorable.

Young leaves and fruits are much more readily infected than older and more mature structures, but late infections are the regular thing in some environments, especially with abundant rain or fog and moderate temperatures. Even apples, especially later maturing varieties, that were apparently free from scab at the time of picking, but were taken from scabby orchards, may develop infections after they go into storage. An extreme case has been reported in which Stayman and Rome Beauty,

picked and stored during the first three weeks of October, showed 80 to 90 per cent with storage type of lesions (Bratley, 1940).

While new infections from the ascospores are the most frequent origin of the first spring lesions, there are certain environments or seasonal conditions when the first infections may come from conidia produced on overwintering twig lesions. This is true in some regions (English records) where twig infections are numerous and ascospores do not reach maturity until after the first spring infections have developed.

The time of initiation of ascospore expulsion and the repetition of the process have a very important bearing on control. While it is a common thing to find abundant discharge of spores about the time the blossoms show pink, activity may begin earlier or it may even be delayed until after the blossoms have opened. In regions or seasons of early spore expulsion, scab is likely to be more severe, and epiphytotics are more frequent. Some attempts have been made in certain commercial areas to maintain a centralized spray service to notify growers when to spray on the basis of maturity and possible expulsion of ascospores, the stage of the fruit buds and the probable weather conditions. Studies on ascospore expulsion seem to have been a favorite pastime for many plant pathologists the world over, and wide variations have been revealed even in regions near together.

Predisposing Factors.—The abundance and severity of scab in any locality are dependent on the proper combination of climatic factors, especially temperature and moisture, which influence the development and expulsion of ascospores, the primary infections by ascospores and secondary infections by conidia. The temperatures of January, February and March are believed by some to be more important to ascospore development than the amount of precipitation in the period immediately preceding the maturity of the ascospores. The optimum for ascus and spore formation has been given as 17°C., with good development at 13 to 21°C. up to a maximum of 29°C. Germination of ascospores or conidia may occur from 0.5 to 32°C. with infection resulting at temperatures from 6 to 26°C. Continuous wetting of surfaces for a time is necessary for infection, the period of wetting required being longer for the low than for the higher temperatures. When spores are prevalent, a cool rain followed by conditions which favor the retention of moisture, or showers in the evening followed by a calm night are very favorable to infection. High spring temperatures, bright sunshine, and little rain are unfavorable for scab, and probably have excluded the disease from important apple districts of Washington.

Scab is increased in severity by: (1) poor pruning resulting in dense branching and heavy foliage; (2) planting in low pockets where the air drainage and evaporation are retarded; (3) by overstimulation of the

trees by nitrogenous fertilizers; and (4) by cloudy weather with low light intensity.

Host Relations and Varietal Resistance.—Many *Pyrus* species of the *Malus* group are susceptible to *Venturia inaequalis*, but scab of pears, hawthorns and mountain ash represent other distinct species.

No varieties of apples are immune to scab, but great variation in susceptibility is shown. There is some evidence that susceptibility is influenced by the rootstock. It is found that certain varieties may be resistant one year and heavily scabbed another year, under conditions favorable for scab. It is also to be noted that varieties noted as resistant in one locality may prove very susceptible in another region. Various writers have listed varieties as resistant, moderately resistant, moderately susceptible or susceptible, but it may be noted that the Baldwin, which is generally classed as resistant, has shown as high as 98 per cent scab. No attempt will be made to explain this and many other similar variations. The general reputation of a variety can, however, be taken into consideration in making selections for commercial orchards. Possibly some of these conflicting reports on varietal resistance may be due to physiological differences of the parasite, as it has been shown that apple scab is a complex of numerous strains varying in both morphologic and physiologic factors. Attempts are being made to cross some of the best wild relatives of the apple that are either immune or highly resistant to scab with superior commercial varieties to secure resistance in commercial stock. This work is still in progress and some promising results have been obtained, but most resistant hybrids lack commercial value.

Control.—The following practices are of value, but of unequal importance, with major emphasis on the use of fungicides:

1. *Planting Practices.*—Set trees in solid blocks of the same variety rather than mixed, to facilitate spraying. Avoid pockets or lowlands with poor air drainage.

2. *Sanitary Measures.*—Keep the trees well pruned to avoid dense growth and heavy foliage which excludes sunshine and retards evaporation and thus favors infection. Either clean cultivation or the close mowing of cover crops or weeds is unfavorable to the development of perithecia and ascospore dissemination.

3. *Spraying or Dusting.*—Success depends on the correct timing of the applications, the frequency of the spraying, the thoroughness of the job and the use of proper materials. In general, spraying has been more effective than dusting. Some of the fungicides are merely protective, while others like lime-sulphur are both protective and eradicative, that is, prevent spore germination and also kill the mycelium of incipient infections. Most failures are caused by lack of thoroughness resulting in

only partial coverage or by improper timing.   The number of applications must be suited to conditions which prevail in the environment, varying from the most severe (A) to the least favorable (J) as presented in the following table:

SCHEDULE FOR SPRAYING OR DUSTING FOR SCAB

A	B	C	D	E	F	G	H	I	J	Relative value, per cent
1. Green-tip stage.................	1									
2. Early closed cluster..............	..	2	2	..	2	..	2	..	..	40
3. Open cluster spray..............	3	3	3	3	3	3	..	3		
4. Calyx (⅔ to ¾ petals off).........	4	4	4	4	4	4	4	4	4	40
5. Ten days to two weeks later.......	5	5	5	5	..	5	..	..	..	10
6. Summer sprays at selected time in four weeks or more after calyx....	6	6	..	6	..	..	..	..	..	10

The following are the more important sprays which have been used with the incorporation of the necessary insecticide (lead arsenate): Bordeaux (4–4–50 to 1½–2–50 or 2–10–50 or even weaker); liquid lime-sulphur (1–40 or 1–50); dry lime-sulphur (3–50 or 4–50); dry-mix lime-sulphur; and clacium sulphide, (8–50 to 12–50).

Repeated experiments have shown the superior fungicidal value of Bordeaux over lime-sulphur for scab control, but because of less injury to foliage and fruit, lime-sulphur has come to be generally used in America, except where lime-sulphur injury is extreme or where other diseases require a copper spray.   Many different proprietary brands of sulphur sprays including wettable sulphurs, colloidal sulphurs, flotation sulphurs, Kolofog and others have given results very comparable to the standard lime-sulphur, especially for later applications.   Coposil, a proprietary copper fungicide (copper ammonium silicate), designed to lessen spray injury, in limited trials has equaled Bordeaux for protection, while Bordeaux injury has been lessened also by a cottonseed-oil Bordeaux according to English investigators.

The following dust mixtures have given the most general satisfaction: (1) dusting sulphur 85 to 90 per cent, plus 10 per cent fluffy lead arsenate; (2) dusting sulphur 40 per cent, lead arsenate 20 per cent and 40 per cent hydrated lime as a filler; (3) hydrated lime 86 per cent, dehydrated copper sulphate 10 per cent and calcium arsenate 4 per cent (Sander's dust). It is now generally conceded that dusting is somewhat inferior to spray-

ing, although better tests and improved dusters have given improvement over the earlier trials.

Spraying to prevent the formation of the ascigerous fruits has been suggested and some success reported. In some of the tests perithecial development has been prevented by late fall applications of 1 per cent helion or 8 per cent fruit-tree carbolineum, while the latter used in the spring is reported to destroy the perithecia in the dead leaves on the ground. Unoiled calcium cyanamid applied at the rate of 20 kilograms per 100 square meters to leaves and soil prevented the development of the perithecia, but this quantity supplies too much nitrogen. Many other compounds have been tried, either before the fall of the leaves or on the fallen leaves. Very promising results have been obtained by the use of Elgetol Extra, a proprietary product containing 13.4 per cent of the sodium salt of dinitro-o-cresol, plus a penetrating agent, applied by a 1 per cent solution by volume at the rate of 450 gallons per acre. By this treatment the ascosporic inoculum has been reduced by 96 per cent.

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BLACK ROT, CANKER AND LEAF SPOT
Physalospora malorum (Berk.) Shear

This is a disease of apples, pears, quinces and numerous other woody hosts, but best known because of its ravages in apple orchards of the eastern United States. The several phases are: a characteristic *leaf spot*, *blight* and *canker* of twigs and limbs, and a *fruit rot* in late maturity or in storage.

The fruit-rot phase was first recognized by Peck in New York in 1879, but the canker phase was not established until 1898, when it was shown by Paddock to be common in New York and adjacent territory. The leaf-spot phase of the disease was not recognized until later, since accompanying fungi (Phyllosticta species) were present and confused the picture. Evidence began to accumulate that the Phyllosticta species were not active parasites, but it remained for Scott and Rorer (1908) to prove definitely by inoculations that the common leaf spot as it occurred in the Middle West was due to the same causal agent as the fruit rot, twig blight and cankers. The ascus stage was first studied by Hesler (1912) when it was classified as a Physalospora, and this was confirmed by later studies of other workers (Shear, 1914, 1924).

Black rot is widely distributed in America from the Gulf States northward to Ontario, Quebec and Nova Scotia, but is rare west of the Rocky Mountains. The canker has been prominent in New York and adjacent territory, while the fruit rot and leaf spot have been the phases general in the extreme East, the Ozarks and Virginia. The disease occurs also in England and continental Europe, South Africa, Australia and New Zealand.

Symptoms and Effects.—The leaf-spot phase of the disease begins soon after the leaves unfold and is first evident as small purple specks

which soon enlarge to circular spots, 2 to 10 millimeters in diameter (average 4 to 5). Later the spots become brown, and when still older as the dead tissue dries out they may assume a dirty gray color. Minute black fruiting bodies (most saprophytic intruders) may be seen occupying the upper surface of mature or old spots. The above description is

Fig. 86.—Apple leaves showing leaf spots caused by black-rot fungus (*Physalospora malorum*). (*After Scott and Rorer, Bur. Pl. Ind. Bul.* **121**.)

typical of the leaf spot that predominates in many localities, but in other regions, especially the southeastern United States, the spot may begin to enlarge or spread and form "brown crescents much darker than the older portion of the spot." These finally fuse, thus making a complete circle of brown around the gray center, and the process is repeated, until a series of concentric circles of brown is formed, giving the condition which has suggested the common name of "frogeye." These lesions may reach a maximum size of 1 inch, under the most favorable conditions. The leaf lesions may be few in number, or numerous, and adjacent spots may fuse to form more extended dead areas.

Localized *cankers* or a *blight* or *dieback* may characterize the attack on trunk, larger limbs, small branches or even twigs. Typical cankers on the large limbs, most frequent on their upper surfaces, are first in evidence as reddish-brown discolorations of the bark, which later become slightly sunken. Some lesions may cease to extend at the end of the first season, while others continue to enlarge year after year until girdling has been completed. At the end of the first year of infection a crevice or crack may be formed at the margin of the lesion, but with

continued advance a series of concentric crevices may be developed. At first the dead bark is closely appressed, but in old lesions it may crack and fall away to some extent. Minute, black, fruiting pustules may be very abundant over the bark of affected twigs or on the cankers. Cankers may be superficial or may extend to the cambium and cause a stain· ing and cracking of the underlying wood.

The *fruit rot* is not in evidence until a few weeks before maturity or later in storage. Fruit lesions are first apparent as small brown spots, frequently at a worm hole, which later darken, expand in size and finally become black. A single lesion to an apple anywhere on the surface is the common condition, but this is frequently located at the calyx

Fig. 87.—Normal apple and shriveled black-rot mummy.

end. No zonation is evident in many cases, but sometimes the lesions may show concentric zones of different shades of brown or black. A fruit lesion continues to advance and to penetrate the flesh until the entire fruit is involved; the affected fruit shrivels and develops a wrinkled surface; and typical black fruiting pustules may be formed. The final result is a dry mummy which may hang on the tree for a time or fall to the ground. The fruit phase of the disease has suggested the very appropriate name of *black rot*.

The injury from the disease is due to the following: (1) the reduced photosynthetic capacities of the spotted foliage and the early defoliation when spotting is severe; (2) the rotting of the fruit just previous to maturity and during storage; (3) the interference of cankers with the life of the branches or girdling of branches and the resultant death of all distal parts; and (4) the blighting or dieback of young affected twigs. In severe cases defoliation may occur six weeks to two months before maturity of the crop, causing the fruit to remain small and of poor quality or to drop prematurely. Such foliage losses make a heavy drain on the

vitality of the tree and seriously interfere with the production of the next year. The amount of fruit rot is variable in different environments, and the injury from the cankers may be scarcely noticeable, or it may be so severe as to cause extensive killing or a reduction in productiveness. The extent to which the decline of trees from old age or from winter injury has played a part seems to have been largely overlooked, the presence of the black-rot organism being taken too often as the indication that it was responsible for the damage.

Fig. 88.—Sections through several types of pycnidia of the black-rot fungus. (*After Leva Walker, Neb. Rept.* **22**, 1908.)

Etiology.—This disease is caused by one of the ascomycetous sphere fungi, *Physalospora malorum* (Berk.) Shear, which produces its ascigerous or perithecial stage in the bark of old cankers, or on affected branches, and its pycnidial or Sphaeropsis stage on the bark, in rotting fruit and more rarely in the leaf spots and on fallen leaves. The rarity of the pycnidial fruits in the leaf spots and the common occurrence of the pycnids of Phyllosticta or other intruding fungi were for many years the source of misconceptions as to the true nature of the disease. The pycnidia are developed in abundance in the bark and on the rotted fruits,

but the perithecial stage seems to be rare, especially in America. It has been proved by single ascospore cultures that these isolations will produce typical pycnidia of the *Sphaeropsis malorum* type.

The septate mycelium is intercellular and is at first hyaline, but soon darkens and becomes greenish yellow, bluish green, brown and then dark brown, appearing black in mass. Sclerotia-like bodies have been observed in cultures and beneath the skin of affected apples. Chlamydospores have been noted in cultures as thick-walled, granular cells, occurring singly, in chains or in groups. Knots of hyphae become aggregated just beneath the outer surface of host parts and gradually develop into the pycnidia or conidial fruits.

FIG. 89.—Section of a perithecium of *Physalospora malorum*. (*After Hesler, Cornell Univ. Agr. Exp. Sta. Bul.* **379**.)

The typical pycnidia are globose, subglobose, elongated or flask-shaped, black or carbonous, immersed in the host tissues or becoming somewhat erumpent, ostiolate, and with distinct but variable outer and inner walls, the inner wall being of thin-walled, hyaline cells, while the outer is of dark, thick-walled cells. They are usually distinct but may be confluent or united into a stroma. The single pycnids are 200 to 300 μ in diameter and produce short, hyaline, clavate or cylindrical conidiophores which arise from the inner face of the hyaline inner wall. Each conidiophore can develop a single, oblong-elliptical, brown spore 7 to 16.2 by 16 to 36 μ. The typical mature spores are brownish and continuous, but they vary from hyaline to very dark brown and from continuous to one-septate, or more rarely to two- or three-septate.

The perithecia occur on twigs and cankers and are very similar in form and size and in character of the wall to the pycnidia. They are immersed in the cortical tissues and protrude by a short papillate ostiole.

The single fruits are 180 to 324 μ high by 300 to 400 wide and produce club-shaped asci, 21 to 32 by 130 to 180 μ, interspersed with hyaline, continuous paraphyses. The two to eight ascospores are ellipsoidal or often inaequilateral, hyaline or greenish yellow, irregularly biseriate, measure 10.8 to 15.2 by 24.4 to 34.2 μ, and are forcibly expelled.

The pathogen may be carried over the winter in the form of dormant mycelium, immatur pycnidia, mature pycnidia, developing perithecia and possibly by pycnospores that have been set free and are lodged on the surface of the bark. Because of the rarity of the perithecia, it seems that ascospores play a very minor role in the life history, main reliance being placed on the pycnospores for the dissemination of the fungus. The pycnospores accumulate in the pycnidial cavity and surrounded by a gelatinous matrix are pushed out as short tendrils, the matrix of which may be dissolved, thus liberating the spores. Rains and insects may bring about their further dissemination, and it has been shown that frog eye infection is correlated with periods of rainfall.

The pycnospores are prevalent and ready to produce infections when conditions are favorable. They have been shown to retain their viability for two or more years and may be liberated during warm, moist periods at any time in the year. The minimum temperature for their germination is 9 to 10°C., the optimum 20°C., the maximum 25 to 30°C. with light a necessary stimulus. Most of the foliage infection takes place from the blooming period to 2½ weeks after the petals fall, that is, while the leaves are still growing. It has been experimentally demonstrated that the pathogen can infect uninjured leaf tissues and produce leaf spots. In some localities, fruit infection independent of injuries has been observed at the calyx end, but various types of injuries to the fruit or bark such as insect injuries, fungous or bacterial lesions, mechanical wounds or frost cankers offer more certain avenues of entrance.

Host Relations.—The disease is primarily a trouble of pomaceous fruits, being of first importance on apple, and of minor importance on crab apple, pear and quince, primarily on the first or the latter. In addition it is found as a saprophyte or as a weak parasite on a considerable number of trees and shrubs.

Variation has been noted in the susceptibility of varieties of the apple. The fruit rot is more severe on early varieties previous to maturity, while late or winter varieties are likely to suffer in storage. The leaf spot and frogeye are reported as severe on Ben Davis, Winesap, Arkansas, Baldwin and Jonathan in Virginia, on Ben Davis in Nebraska, on York Imperial and Stayman Winesap in Pennsylvania and on Chenango, Baldwin, Rhode Island and Twenty Ounce in New York. The leaf spot is much worse in old orchards, particularly where pruning has been neglected, than in young orchards. The Twenty Ounce is most

severely affected by canker and is often killed, while the Esopus has run out in New York because of its extreme susceptibility. Physiological strains have not been definitely established, but different isolations have been shown to vary greatly in rapidity of rot produced and in their behavior in cultures.

Control.—As a basis for prevention or reduction in the amount of the disease the following features should be kept in mind: (1) the pathogen is a wound parasite on limbs and to a great extent on fruits; (2) the fungus can penetrate the unbroken epidermis of leaves to produce leaf spot or frogeye; (3) sporulating pycnidia may be formed on cankers, blighted or dead twigs, fallen spotted leaves and old mummies; (4) the ascigerous fruits on old cankers may play a part in the spread of the pathogen.

Preventive or control measures may be grouped as follows: (1) Prevention of wounds or their protection. Avoid injuries from machinery or equipment used in cultivating, spraying, pruning or picking, and give attention to the control of insect pests and other bacterial or fungous diseases. Pruning wounds or other mechanical injuries that cannot be avoided should be treated with either coal tar or a Bordeaux paint. (2) Cankers of whatever origin should be either treated or the affected limb removed and destroyed. In general large cankers may be treated, while small or unproductive cankered limbs and blighted twigs should be removed and destroyed by burning. (3) The mummies that are hanging on the tree or lying on the ground should be collected and destroyed, and fallen leaves may be removed or plowed under previous to the blossoming period. (4) Spraying is of doubtful value in the prevention of canker but has been used effectively for the prevention of fruit rot and for the control of leaf spot or frogeye. It is generally necessary to spray for scab in those regions in which the leaf-spot phase is prevalent, and the usual experience has been that the disease is controlled by the scab sprays, the pink, calyx and one later spray generally being sufficient to secure practical control, while still further reduction will follow a fourth application, especially if the rains continue. Either lime-sulphur (1 to 25 or 30) or Bordeaux (4–5–50) has been used, but on account of Bordeaux injury to both foliage and fruit, lime-sulphur would seem more desirable unless bitter rot and blotch must also be controlled.

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BLIGHT OR ENDOTHIA CANKER OF CHESTNUT

Endothia parasitica (Murr.) And. and And.

This is a virulent disease which invades the bark and cambium of twigs, branches or main trunk, forming cankers that ultimately girdle these structures and cause the death of all distal parts, thus producing blight or dieback of twigs or branches or leading to the death of the entire tree. The disease has been called the chestnut-tree blight or the chestnut bark disease, but Endothia canker has been used more recently. Since a typical blight is not produced except on the small twigs, and other cankers of the chestnut are known, and since both bark and wood are affected, it might be best to adopt the specific name of *Endothia canker*.

Since the first record of the disease in the New York Zoological Park in 1904, it spread with alarming rapidity from this original center. It became serious in states to the East, and to the West and Southwest, and by 1911 the rapid advance in the important chestnut forests of Pennsylvania led to the establishment of the Pennsylvania Chestnut Tree Blight Commission with an appropriation of $275,000 to study the possibility of eradication or of checking its spread. The study was also facilitated by cooperation with the Federal Office of Forest Pathology, which continued the study after the Commission ceased in 1913. The publication of a bibliography of the disease in 1941 contains 399 titles, which gives emphasis to the importance of the epidemic. After 1914 general control measures were discontinued for the most part and the disease continued to extend its ravages, until it is now prevalent throughout the natural range of the chestnut and has occurred on cultivated chestnut as far west as California, Oregon, Washington and British Columbia.

It was first reported from Europe on the cultivated species in 1924. At first the causal fungus was thought to be native to America, but it was discovered in China in 1913 on the native Chinese chestnuts (*Castanea mollissima*) and later in Japan on native wild trees (*C. crenata*). These discoveries gave rather definite proof that the pathogen is an importation from one of these countries.

Symptoms and Effects.—The appearance of infections will be different, depending on the age of the twigs, shoots, limbs or trunk. Young infections on smooth-barked, vigorous shoots show as yellowish or yellowish-brown, slightly raised patches, in contrast to the olive-green healthy bark. These lesions may show a regular outline or sometimes a very irregular margin, or an amoeboid form, but in either case there are no fruiting pustules until later. The mycelium or vegetative body of the fungus, whitish or buff-colored, is readily shown by the removal of the

brown cork cells from the advancing edge of the lesion. These lesions may spread until the shoot is completely girdled, when fruiting pustules may be formed.

Young infections on slow-growing twigs or on the smooth bark of older branches or trunks are not so evident, but they generally show as somewhat discolored, dead areas, sometimes slightly depressed and occasionally with a raised margin. The lesion may be nearly circular or target like, but it is generally elongated in the direction of the long axis of a shoot or branch and continues to enlarge until the organ is

FIG. 90.—Chestnut limbs showing cankers due to *Endothia parasitica*.

completely encircled. Small shoots may be girdled before the appearance of the fruiting pustules, but these may form on the larger limbs or main trunk before the girdling has been completed. The fruiting bodies show as small, yellow, orange or reddish-brown pustules which break through the bark some distance back from the advancing edge of the lesion.

Young infections on old trunks or large limbs with thick fissured bark cause little change in the appearance of the bark itself, and the fungus may have gained considerable headway before there is any external evidence of its presence. Sometimes the first indication of an infection on large limbs or trunk is the formation of abnormal longitudinal splits or fissures, and the orange or yellow fruiting pustules may soon appear in the deep cracks or crevices.

On any of the three types of lesions just described, during damp weather following rain periods or in moist situations, long, irregularly twisted, buff or bright yellow threads may be extruded from some of the pustules. These tendrils or spore horns are masses of conidia or summer spores, soft and sticky at first, but hard and brittle when dry.

Some infections, especially those on vigorous shoots, may cause a pronounced enlargement or hypertrophy, which may involve the entire lesion or only the upper portion. Longitudinal splits or fissures in the bark are characteristic of hypertrophied lesions. Sometimes a lesion may show a marked sunken area due to the killing of the invaded bark,

FIG. 91.—Piece of an infected branch showing numerous pycnidia and extruded spore coils or "spore horns."

while the bordering tissues have grown at a normal rate. A typical canker may thus result with cracked and fissured bark, and in old lesions in which girdling has been completed, the bark may begin to peel away from the wood.

Aside from the discovery of the actual lesions there are other signs that indicate the presence of blight: (1) dead leaves hanging in characteristic drooping clusters, which generally remain on the tree during the winter; (2) the persistence of burrs, frequently undersized, on the blight-killed branches; (3) limbs bearing normal or dwarfed leaves which become chlorotic, reddish-brown or brown and finally fall leaving these shoots defoliated; (4) the production of clusters of vigorous, rapid-growing sprouts localized on branches, trunk or crown, just below a girdling lesion.

The final effect of cankers is to kill the parts beyond the girdled zone. Lesions on the main trunk are especially serious, since with the completion of the girdling, the entire top must succumb. If the base of the trunk and crown are not invaded, new shoots may develop. Top infections for several years may cause the death of groups of branches or a staghead effect. The wood of blight-killed trees is injured but little as a direct result of the disease, but if left standing it soon begins to deteriorate as a result of the work of insects and various species of wood-destroying fungi.

Etiology.—This chestnut disease is caused by *Endothia parasitica* (Murr.) And. & And., one of the sphere fungi (Sphaeriales) which produces a pycnidial stage to be followed later by stromata-bearing peri-

FIG. 92.—Section through a single pycnidium.

thecia. The disease can be readily transmitted by inoculations made from pure cultures or by the transfer of mycelium from active lesions. It is, however, strictly a wound parasite, being unable to penetrate the unbroken periderm of either young or old parts. The mycelium develops principally in the living cambium and bark and to a limited extent in the outer sapwood of any part above ground and continues to grow in dead parts as long as moisture and food are available. The mycelium is at first a cottony white, but soon becomes buff or yellowish and spreads out in the bark layers or in the cambium in the form of closely appressed sheets or fanlike layers.

Following an internal development of vegetative hyphae, the mycelium forms *pycnidial pustules* or stromata which break through the bark as small, yellow or orange-colored papillae. Each pycnid is a dense aggregate of fungous tissue, generally containing one (rarely more) large, lobulated cavity, lined with innumerable vertical filaments or conidiophores, which give rise to enormous numbers of hyaline, continuous, cylindrical, straight or slightly curved pycnospores, 1.28 μ wide

to 3.56 μ long.   The accumulation of the pycnospores causes a rupture
of the pycnidial wall, and the spore mass oozes out in the form of a

FIG. 93.—Perithecial stromata in the crevices of rough bark.

FIG. 94.—Groups of perithecial stromata somewhat enlarged.

yellow or orange, threadlike or flattened, irregular coil, the so-called
"spore horn" or tendril.   These spore horns are produced during humid
periods following rains and persist until washed away during some later

storm, but during rains the pycnospores are washed away as fast as they are forced to the surface. A single spore horn of average size may contain many million (115) pycnospores.

After a period of activity the pycnidial pustules may be transformed into perithecial stromata, which then show upon their surface a number of raised papillae or a number of minute black dots, the ostioles or openings of the perithecia or flasklike bodies buried deep in the stromata.

Fig. 95.—Section of a perithecial stroma showing immersed, flask-shaped perithecia which open to the surface by long necks.

Each stroma may contain 1 to 60 (average 15 to 30) distinct flasklike perithecia, each of which opens to the surface by a long black neck. The outer body of the perithecium consists of 10 to 12 layers of compact, dark, heavy-walled cells, surrounding the inner wall of two to three layers of thin-walled cells from which the asci originate. The neck of a perithecium is lined with thin-walled hyphae which project inward and upward and are especially prominent near the ostiole. The asci are oblong or broadly clavate, eight-spored, average 8.9 by 51.2 μ, and have a very delicate hyaline wall with a thickened ring at the apical end. The ascospores are hyaline, oblong or oval, one-septate, generally constricted at the septum, average 4.5 by 8.6 μ and are arranged in one or two irregular rows.

When mature perithecia are sufficiently moist and the temperatures are favorable, the asci are detached from the wall, and as they accumulate are forced into the long neck in a linear series. When an ascus reaches the ostiole and its tip is exposed to the exterior, it explodes and the eight ascospores are projected into the air. Another ascus is pushed up, expels its spores, and the process continues, so each perithecium is really a repeating spore gun. The ascospores by their method of discharge are adapted to a wind dissemination, while the pycnospores are washed down by rains.

The pycnospores are produced in abundance at all times of the year when temperature and moisture conditions are favorable and are washed down in large numbers from diseased branches even during warm winter rains, when spore horns are rarely observed, but in greater numbers during the rains of spring and summer. They remain viable for days on normal bark below lesions and are able to survive two to three months in perfectly dry soil; they are carried in large numbers on the bodies of insects and birds that frequent cankers. Pycnids may even be formed on the bare wood of peeled logs, on the cut ends of the

Fig. 96.—Asci showing form and position of ascospores.

sapwood or even on fragments of bark or wood left in chopping. Rain, insects and birds are the important agents of pycnospore dissemination and, considering their resistance to desiccation, pycnospores might even be transported long distances on the bark of healthy nursery trees or in soil adhering to their roots.

Fig. 97.—A comparison of maximum and minimum sizes of ascospores and pycnospores.

Maturing perithecia may be found at any season of the year, since their development depends more upon the age of the lesion than upon the season, but they may be more abundant in the fall and winter than at other seasons. The perithecia may continue to be formed on the bark of blight-killed trees or on the bark of fallen logs. The drying of the bark does not prevent the perithecia from expelling spores when again subjected to favorable moisture and temperature conditions. The expulsion of the ascospores takes place under natural conditions only when the stromata have been moistened by rains, but before they have dried. Analytic tests have shown them to be very prevalent in the vicinity of

infected chestnuts during the first 5 hours following the cessation of a rain, but less abundant later, sometimes for even 14 hours after the rain. Only a few spores are expelled at 54°F., the number increasing as the temperature rises, with the maximum expulsion occurring at 68 to 80°F. This means then that expulsion begins with the first warm rains, increases as the conditions become more favorable, then declines in the fall when lower temperatures prevail and ceases entirely during the cooler portions of the year. The activity of pustules is not exhausted in a single season but may be as vigorous during the second season as during the first.

By means of exposure plates and spore traps, it has been shown conclusively that the ascospores are wind-disseminated, being carried away from diseased trees in large numbers following each warm rain of any appreciable amount. They have been obtained in large numbers from the air 300 to 400 feet from the nearest source of supply; hence they must be carried much greater distances.

It has been proved by experimental tests, using either pycnospores or ascospores, that the fungus cannot enter the normal bark, and even active, vegetative mycelium cannot penetrate. Injuries of some sort are necessary to open the bark. These may be made in a variety of ways, but it is believed that certain insects, for example, a bast miner, are the most important agents in making openings.

Economic Importance.—The completeness of destruction wrought by the disease is without parallel in the annals of plant pathology. It has been making a clean sweep, killing young growth and merchantable timber of the forests, the planted groves for nut production, and the beautiful shade trees of cities and country estates. In the areas of earlier infestations the fine chestnut forests, groves and shade trees have entirely disappeared, and it seems possible that the extermination of the chestnut throughout its natural range may be the final result. All this destruction has happened because a struggling and insignificant parasite was carried from its Oriental home to a new environment.

Host Relations.—All varieties of chestnuts, including the native American species, *Castanea dentata,* and the European and Japanese species are susceptible to Endothia canker. The eastern chinquapin (*C. pumila*) and the western chinquapin (*Castanopsis chrysophylla*) can also contract the disease, but show considerable resistance. It is unfortunate that the native American species, the most valuable species commercially, should be so exceedingly susceptible. The breeding problem has for its ultimate goal the production of resistant varieties for nut production and other varieties that may be grown as timber trees. Some progress has been made in the production of resistant horticultural varieties by hybridizing and by the introduction of resistant species and strains from the Orient. The canker fungus can grow as a saprophyte

on the bark of various trees and has been found growing naturally on several species of oaks, *Acer rubrum, Carya ovata* and *Rhus typhina,* but it is so weak a parasite on these species that it is not a menace.

Control.—The control of Endothia canker has been attempted under strictly forest conditions, in nut-producing orchards and in ornamental shade trees with but little success in any case. Early in the epiphytotic, careful pruning, tree surgery or the cutting out of cankers alone or combined with spraying were used in attempts to save valuable shade and orchard trees, but despite the most painstaking efforts owners saw their prized trees gradually go down. Injections of toxic chemicals into the bark resulted in neither protection nor cure.

The possibility of limiting the state- or nation-wide spread of the disease through the native forests was given special attention by Federal and state authorities. The most complete tests by the Pennsylvania Chestnut Tree Blight Commission failed to do more than slightly retard the advance of the disease, and since that time only sporadic efforts have been made by either state or Federal workers.

Within the invaded forest areas the problem has become one of the utilization of the chestnut timber in the most efficient way. Blight-killed timber deteriorates quite rapidly, hence should be cut and marketed as soon as possible, and the devastated areas devoted to other desirable species.

The only hope for the chestnut is the breeding of resistant or immune varieties (see Host Relations). The extermination of the American chestnut throughout its range seems certain unless nature herself intervenes. Extermination of species of trees has characterized former geologic times, so it may be that such a process is being enacted before our eyes at the present time.

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IMPORTANT DISEASES DUE TO ASCOMYCETES

For Key references on these diseases see F. D. Heald, "Manual of Plant Diseases," 2d ed., pp. 517–518; 560–562; 583–586; 656–663.

EXOASCALES (*Exoascaceae*), pp. 517–518

Principal host	Common name of disease	Scientific name of causal organism
Plum............................	Plum pockets, "bladder plums," "fool's"	*Taphrina pruni* (Fcl.) Tul *T. communis* (Sad.) Gies.
Cherry............................	Witches'-broom	*T. cerasi* (Fcl.) Sad.
Cherry............................	Curl	*T. minor* Sad.
Peach............................	Leaf curl	*T. deformans* (Fcl.) Tul.
Pear............................	Leaf blister	*T. bullata* (Fcl.) Tul.
Oaks............................	Leaf blister or curl	*T. coerulescens* (M. and D.) Tul.

HELVELLALES, p. 560

Conifers........................	Root rot	*Rhizina inflata* (Schäff.) Sacc.

PEZIZALES, pp. 560–561

Stone fruits........................	Brown rot	*Monilinia* spp.
Lettuce and garden vegetables........	Drop or wilt	*Sclerotinia sclerotiorum* (Lib.) Mass.
Lemon............................	Cottony rot	*S. sclerotiorum* (Lib.) Mass.
Lettuce and garden vegetables........	Drop or wilt	*S. minor* Jagger
Castor bean........................	Gray mold	*S. ricini* Godfrey
Clover and alfalfa..................	Stem rot or wilt	*S. trifoliorum* Eriks.
Cranberry........................	Hard rot and tip blight	*S. oxycocci* Wor.
Larch............................	Canker	*Dasyscypha calycina* (Schum.) Fcl.
White pine........................	Canker	*D. fusco-sanguinea* Rehm
Alfalfa............................	Leaf spot	*Pseudopeziza medicaginis* (Lib.) Sacc.
Currant and gooseberry.............	Anthracnose	*P. ribis* Kleb.
Grape............................	*Roter Brenner*	*P. tracheiphila* MT.
Apple, pear, etc	Black spot canker	*Neofabraea malicorticis* (Cord.) Jack.
Apple, pear, etc	Perennial canker	*Neofabraea perennans*(Z.&C.) Kienh.
Pear and quince....................	Leaf blight	*Fabraea maculata* (Lev.) Atk.
Alfalfa............................	Yellow leaf blotch	*Pyrenopeziza medicaginis* Fcl.
Pine and fir........................	Twig blight	*Cenangium abietis* (Pers.) Duby

PHACIDIALES, p. 561

Arborvitae........................	Black leaf spot	*Keithia thujina* Dur.
Cherry............................	Leaf spot	*Coccomyces hiemalis* Higgins
Plum............................	Leaf spot	*C. prunophorae* Higgins
Poplar............................	Anthracnose	*Trochila populorum* Desm.
Maple............................	Tar spot	*Rhytisma acerinum* (Pers.) Fr.
Douglas fir........................	Needle blight	*Rhabdocline pseudotsugae* Syd.
Apple and pear....................	Canker and fruit rot	*Phacidiella discolor* (M. and S.) A. Pot.

HYSTERIALES, p. 562

Principal host	Common name of disease	Scientific name of causal organism
Larch	Leaf cast	*Hypodermella laricis* Tubeuf
Western yellow pine	Leaf cast and witches'-broom	*Hypoderma deformans* Weir
White pine	Leaf cast	*H. strobicola* Tubeuf
Pine and fir	Leaf cast	*Lophodermium pinastri* (Schr) Chev.

PERISPORIALES, pp. 583–586

Apple and pear	Blue-mold rot	*Penicillium expansum* Link
Citrus fruits	Blue-mold rot	*P. italicum* Wehmer
		P. digitatum Sacc.
Fig and date	Smut	*Aspergillus niger* Van Tiegh.
Onion	Black mold	*A. niger* Van Tiegh.
Rubus spp	Anthracnose	*Plectodiscella veneta* (Speg.) Burk.
Onion	Smudge and black spot	*Cleistothecopsis circinans* (S. and T.)
Orange	Sooty mold	*Meliola penzigi* Sacc.
Rose, strawberry, etc	Powdery mildew	*Sphaerotheca humuli* (DC.) Burr.
Peach, rose, etc	Powdery mildew	*S. pannosa* (Wallr.) Lev.
Gooseberry	Mildew	*S. mors-uvae* (Schw.) (B. and C.)
Pea, clover, etc	Powdery mildew	*Erysiphe polygoni* DC.
Cereals and grasses	Powdery mildew	*E. graminis* DC.
Cucurbits, composites, etc	Powdery mildew	*E. cichoracearum* DC.
Grape	Powdery mildew	*Uncinula necator* (Schw.) Burr.
Apple, pear, quince, etc	Powdery mildew	*Podosphaera leucotricha* (E. and E.) Salm.
Cherry	Powdery mildew	*P. oxyacanthae* (Fries) DeB.
Oak	Mildew	*Microsphaera quercina* (Schw.) Burr.
Trees and shrubs	Common tree mildew	*Phyllactinia corylea* (Pers.) Karst.
Rose	Black spot or blotch	*Diplocarpon rosae* (Fr.) Wolf.
Strawberry	Leaf scorch	*Diplocarpon earliana* (E. and E.) Wolf.

HYPOCREALES, pp. 656–657

Apple, pear, etc	Canker	*Nectria galligena* Bres. et al.
Various woody species	Dieback or coral spot	*N. cinnabarina* (Tode) Fr.
Cereals	Seedling blight or snow mold	*Calonectria graminicola* (Berk. and Br.) Woll.
Small grains	Seedling blight, foot disease, scab	*Gibberella saubinetii* (Mont.) Sacc.
Corn	Root, stalk and ear rot	*G. saubinetii* (Mont.) Sacc.
Rye and other cereals	Ergot	*Claviceps purpurea* (Fr.) Tul.
Rice and corn	False smut	*Ustilaginoidea virens* (Cke.) Tak.
Grasses	Cattail fungus	*Epichloe typhina* Tul.
Insects, various	Cast fungus	*Cordyceps militaris* (L.) Link and other species

DOTHIDIALES, pp. 657–658

Clover	Black spot	*Phyllachora trifolii* (Pers.) Fcl.
Grasses	Black spot	*P. graminis* (Pers.) Fcl.
Elm	Black spot	*Dothidella ulmi* (Duv.) Wint.
Plum and cherry	Black knot	*Dibotryon morbosum* (Schw.) T. and S.
Currant and gooseberry	Black knot	*Dibotryon ribesia* (Pers.) Sacc.

SPHAERIALES, pp. 658–663

Principal host	Common name of disease	Scientific name of causal organism
Sugar cane	Pineapple disease	*Ceratostomella paradoxa* (DeS.) Dade
Western yellow pine	Bluing	*C. pilifera* (Fr.) Wint.
Sweet potato	Black rot	*C. fimbriata* Elliott
Stone fruits	Bark fungus	*Calosphaeria princeps* Tul.
Apple	Black rot, canker and leaf spot	*Physalospora malorum* (Berk.) Shear
Stone fruits	Blight	*Ascospora beijerinckii* Vuill.
Stone fruits	Dieback	*Valsa leucostoma* Fr.
Filberts and hazel	Blight	*Cryptosporella anomala* (Pk.) Sacc.
Grape	Dead-arm disease	*C. viticola* (Red.) Shear
Currant	Cane blight	*Botryosphaeria ribis* G. and D.
Apple, pear, quince and grape	Bitter rot	*Glomerella cingulata* (St.) Sp. and von S.
Cotton	Anthracnose	*G. gossypii* (South.) Edg.
Miscellaneous fruit trees and herbaceous spp.	Root rot	*Rosellinia necatrix* (Hart.) Berl.
Oak	Root rot	*R. quercina* Hart.
Hickory	Canker	*R. caryae* Bonar
Cranberry	Rot	*Acanthorhynchus vaccinii* Shear
Poplar	Canker	*Hypoxylon pruinatum* (Klot.) Cke.
Apple	Blister canker	*Nummularia discreta* (Schw.) Tul.
Apple	Black-root rot	*Xylaria mali* Fromme
Oak	Canker	*Diaporthe taleola* Fr.
Sweet potato	Dry rot	*D. batatatis* (E. and H.) H. and F.
Rose	Brown canker	*D. umbrina* Jenk.
Lima bean	Pod blight	*D. phaseolarum* (C. and E.) Sacc.
Chestnut	Blight or Endothia canker	*Endothia parasitica* (Murr) And. and And.
Sycamore	Anthracnose	*Gnomonia veneta* (Sacc. and Speg.) Kleb.
Elm	Leaf spot	*G. ulmea* (Schw.) Thum.
Walnut	Anthracnose	*G. leptostyla* (Fr.) Ces. and d. Not.
Grape	Black rot	*Guignardia bidwellii* (Ellis) V. and R.
Cranberry	Blast or early rot	*G. vaccinii* Shear
Horse chestnut	Leaf blotch	*G. aesculi* (Pk.) Stew.
Strawberry	Leaf spot	*Mycosphaerella fragariae* (Schw.) Lind.
Rubus spp.	Leaf spot	*M. rubi* Roark
Raspberry	Spur blight	*M. rubina* (Pk.) Jacz.
Currant and gooseberry	Leaf spot	*M. grossulariae* (Fr.) Lind.
Pear	Leaf spot	*M. sentina* (Fr.) Scrot.
Cucurbits	Wilt and black rot	*M. citrullina* (Sm.) Gross.
Beets	Dry-heart rot and leaf spot	*M. tabifica* (P. and D.) Johns.
Peas	Blight	*M. pinodes* (B. and Bl.) Stone
Cauliflower	Ring spot	*M. brassicola* (Duby) Lind.
Iris	Leaf spot	*Didymellina macrospora* Kleb.
Apple	Scab	*Venturia inaequalis* (Cke.) Wint.
Pear	Scab	*V. pirina* Aderh.
Poplar	Scab and dieback	*V. tremulae* Aderh. Syn. *Didymosphaeria populina* Vuill.
Pine	Brown-felt blight	*Neopeckia coulteri* (Peck) Sacc.
Conifers	White-felt blight	*Acanthostigma parasiticum* (Hart.) Sacc.
Conifers	Brown-felt blight	*Herpotricha nigra* Hartig
		H. quinqueseptata Weir
Raspberry	Cane blight	*Leptosphaeria coniothyrium* (Fcl.) Sacc.
Oats	Speckled blotch	*L. avenaria* Weber
Wheat	Speckled leaf blotch	*L. tritici* (Gar.) Pass.
Wheat and other cereals	Take-all	*Ophiobolus cariceti* (B. and Br.) Sacc.
Alfalfa and clover	Leaf spot	*Pleosphaerulina briosiana* Pol.
Barley	Stripe disease	*Pleospora gramineum* Died.
Barley	Net blotch	*Pyrenophora trichostoma* (Fr.) Wint.

CHAPTER IX

DISEASES DUE TO BASIDIOMYCETES

SMUT FUNGI, USTILAGINALES

The smut fungi generally form black spore masses (except Entyloma), which either break up into a fine dustlike powder, readily scattered by the wind (loose smuts), or remain firm and more or less covered (kernel or covered smuts). All are obligate parasites.

Hosts and Economic Importance.—The smut fungi of the most economic importance are those affecting the cereals and some of the wild and cultivated grasses. A few other economic plants are also attacked by smuts (see page 188). The economic importance of the smut diseases is due (1) to the world-wide cultivation of cereals; and (2) to the fact that parts of these plants most frequently destroyed by the smuts are either the grains or the entire inflorescence, thus lowering quality and yield of the commercial product.

General Characters.—The mycelium of a smut may grow throughout the tissues of the host from the seedling stage to maturity (systemic type) or the mycelium may be localized in certain aerial parts. The characteristic features of the order are: (1) the formation of single or grouped resting spores (chlamydospores), which generally accumulate in black powdery masses or *sori;* (2) the germination of the resting spores to form either a *promycelium* (indirect germination), or an *infection thread or hypha* (direct germination); and (3) the budding of the secondary spores, or sporidia, in either soil moisture or nutrient solutions to form either yeastlike forms or secondary sporidia of a different type. In a few cases conidia are formed on the surface of affected parts (*e.g., Entyloma spp.*).

In the indirect germination two types of *promycelia* are formed: (1) a two- to four-septate hypha with elliptical or oval, uninuclear *sporidia*, or secondary spores, which bud out from the distal end of each cell (Ustilago type); or (2) a long or short continuous hypha which produces a crown or whorl of sporidia, or secondary spores, from its free end (Tilletia type). The sporidia may germinate to produce an infection thread or hypha which may infect the host, or these sporidia may bud indefinitely in nutrient solutions, and the buds give rise to infection hyphae when conditions are favorable. In the direct germination, a resting spore forms a simple septate hypha, which may penetrate the

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host at once and establish an infection, or, in a nutrient solution, a much-branched septate mycelium may be formed.

Types of Infection.—Three important types may be illustrated by the smuts of our cultivated crops:

1. *Seedling Infection.*—In these smuts, infection can take place only during the young or seedling stage from spores that were either carried on the seed or were present in the soil or in loose smut of oats from mycelia that infected the glumes at flowering time. The bunt or stinking smut of wheat, the loose and covered smuts of oats, the covered smut of barley, kernel smut of sorghum and millet smut illustrate seedling infection from seed-borne spores. Soil contamination may result from *wind-blown smut,* as illustrated by the "smut showers" of the Inland Empire of the Pacific Northwest and some other similar sections, or it may be

FIG. 98.—Various types of smut spores. *A,* single-celled spores, smooth and with various wall markings; *B,* series of spores from two to many celled. Stippled cells are fertile, others sterile.

residual, that is, the spores may originate from a smutty crop grown on the same ground the previous year or even at an earlier time. Residual soil contamination is an important feature of flag smut of wheat, onion smut and head smut of corn and sorghum. In seedling infection, the infection threads which originated from either primary or secondary sporidia enter the young seedling, reach the growing point and then keep pace with the growth of the host tissues until the heads are formed, when the characteristic smut masses appear.

2. *Blossom Infection.*—In the loose smuts of wheat and barley, the infection takes place at blossoming time. The spores from smutted heads, which reach maturity about the time normal heads are in blossom, are blown by the wind, and infect young ovaries of normal heads, producing therein an internal mycelium which is developed within the seed. When the seed starts to germinate, the mycelium keeps pace with the growing points of the young seedling and finally develops in the heads

where the spores are organized and matured. In this type the fungus exists in the "germ," or embryo of the seed, as a vegetative mycelium, ready to resume activity with the awakening seed. Infected seed, that is, seed in which an internal smut mycelium has been formed, if planted the next season, may produce plants affected with the loose smut.

3. *Shoot Infection.*—In the common smut of corn, infection is localized in various aerial parts, that is, there is no systemic infection as in the two other types. Spores may germinate on leaves, stalks or flower parts, and form infection threads which enter the host tissue and establish a mycelium. As a result of these local infections, small or large smut masses or sori may be developed from the purely local mycelium, but there is no general spread of the mycelium within the host tissue from an original center of infection. The spores may be residual in origin or they may be brought into the field from some other source; infection may result from either the primary or secondary sporidia carried by the wind to the parts which they infect.

The smuts are grouped in two families, which are characterized mainly by the method of germination and by the form of the promycelium.

I. Ustilaginaceae

Germination is by means of a septate promycelium with lateral and terminal sporidia or by a septate infection thread which does not form sporidia. Sori generally form naked or covered, exposed, dusty or agglutinated masses, but, in a few cases, the spores may be buried within the host tissue. The spores are continuous, two celled or united in spore balls or groups. The principal genera of economic importance are: *Ustilago, Sphacelotheca* and *Sorosporium.*

II. Tilletiaceae

Germination is by a long or short nonseptate promycelium which forms an apical whorl of elongated or filiform sporidia. These sporidia may fuse in pairs or not and either form infection threads direct or produce secondary sporidia which are either similar or dissimilar. The sori are either dusty and exposed or permanently embedded in the host tissues. The spores are light brown or dark brown (hyaline in *Entyloma spp.*), single, with or without hyaline appendages and either dusty or not readily separating, or in groups or balls. The principal genera furnishing pathogens of economic importance are: *Tilletia, Urocystis* and *Entyloma.*

BUNT OR STINKING SMUT OF WHEAT

Tilletia tritici (Bjerk.) Wint. and *T. levis* Kühn

The common name "bunt" is of English origin, a dialectal contraction of "burnt ears" to "bunt ears" and finally to "bunt," while the pro-

nounced odor has suggested the appropriate name of *stinking smut.* There are two common species of bunt, the rough-spored form *Tilletia tritici* and the smooth-spored form *T. levis.* Bunt of wheat is present to some extent in every country of the world where wheat is grown. A third species, *T. indica,* is of local occurrence on wheat in India.

Stinking smut of wheat was apparently the first smut to attract attention and was undoubtedly known in ancient times. In 1755 Tillet, noted the difference between *la carie,* or stinking smut, and *le charbon,* or loose smut. Smut was at first considered as a degeneration of the grain, and it was not until 1807 that Prevost discovered that the spores germinated in water and so were of fungous origin. The true nature of the fungus was determined by the later studies of Tulasne (1854), Kühn (1874) and especially by Brefeld (1883). During following years the contributions on wheat smut became more numerous, and, even today, the output still rolls on with apparently increasing volume.

Since the recognition of seed contamination as the source of inoculum, investigators of the bunt problem have concerned themselves primarily with methods of control by seed treatment. Seed disinfection by several standard fungicides is now recognized as a dependable means of control in most countries. The failure of these fungicides to give control in some sections, as in the Pacific Northwest, is due to soil contamination by wind-blown spores during the harvest period. Therefore, continued investigations on control of bunt by seed treatment should be directed primarily toward the discovery of some compounds which will be effective in reducing the smut due to soil contamination. In more recent years, emphasis has been placed upon a study of epidemiology, or the conditions which favor the development of the disease, while, in the last few years, the recognition of physiological specialization in both species has brought this phase of the problem into prominence.

Symptoms and Effects.—Bunt is easily detected after the wheat is in head by deviation from normal in the form of infected heads, two types being prevalent: (1) the change of the normally compact or square heads in the "compactum" varieties, such as Little Club, Hybrid 128 and others, to a slender type, even exceeding the normal heads in length; and (2) very slight or quite evident change in the "vulgare" varieties by some divergence of the glumes due to the expansion of the smut berries. Frequently, smutted heads, previous to maturity, are darker green than normal and remain green longer. When the normal grains are in the "dough" stage, the smutted grains are filled with a soft, black pasty mass, but, in the mature grain, the pasty interior of the smutted berries has changed to any oily powder, the characteristic smut mass. In some varieties smutted heads stand erect when normal ones are beginning to droop, while, in bearded varieties, the presence of smut frequently causes

a shedding of the awns. Badly smutted plants may be normal in height or in many cases reduced in size, with either increase or decrease in tillering. Size reduction is more common for *Tilletia tritici*.

Infection on young seedlings is frequently indicated by white or leprous spots on the coleoptile and first leaf and by a twisting or distortion. In certain cases, especially in resistant varieties, infection may

Fig. 99.—Smutted and normal heads of Jones Winter Fife and Hybrid 128. Note the marked change of form of the smutted head of the club wheat.

apparently occur without the final appearance of smut heads. In this so-called "latent" infection some of the effects may be the twisting and distortion of seedlings; general weakness with production of shorter stems and heads; and increased liability to winterkilling.

Infection causes morphological deviations from the normal that are apparent before the smut balls have been completed. A few days before emergence from the boot, smutted plants show: ovaries increased in size and green instead of white; stamens reduced in length and breadth;

and anthers pale yellow instead of green, reduced in size, and with imperfectly organized pollen cells.

The effect of the smut fungus upon its host may be summarized as follows: (1) the consumption of food, since the smut fungus must live at the expense of its host; (2) the stimulating or retarding effect on normal physiological processes; and (3) the destruction of the seed or grain in the sporulating process, leaving nothing but the brown outer seed layer (pericarp) enclosing the mass of smut spores. A plant may be wholly or partially smutted, that is, the heads from a single stool may be all smutted, or some normal and some smutted; heads may be completely smutted or only partially smutted, with the minimum degree represented by a single partially smutted berry; while individual berries may be all smut, or the mass may occupy only a small portion of the grain. In general, the more resistant the variety, the greater the number of partially smutted plants and partially smutted heads. Under abnormal conditions in greenhouse cultures, smut masses have been developed as wartlike galls on the leaves or even in the stems.

Losses from Bunt.—The injury or financial loss to be charged to the bunt account is fourfold: (1) increased cost of production due to seed treatment, soil sanitation and cultural practices designed to reduce infection; (2) the reduction in yield per acre; (3) the lowering of grade or quality; and (4) the losses from separator and grain fires caused by smut explosions.

Seed treatment involves much extra labor, the use of special apparatus or machines and enormous quantities of fungicides, sometimes an increased amount of seed and, in certain cases, causes poor stands or even complete failure owing to the killing effect of the fungicide employed. The production of smut in a field follows the use of smutty seed without treatment, the use of improper or poorly applied fungicides and will vary with cultural and planting practices in those areas in which there is a soil contamination. Bunt causes reductions in yields depending upon the percentage of the heads that are smutted. With few smutted plants, possibly up to 5 per cent, the yield reduction is negligible or slight, since the normal plants crowd the smutted ones, but, with high degrees of smutting, it comes to more nearly equal the per cent of smutted plants. In actual farm practice, the amount of smut may vary from traces to as high as 75 to 80 per cent if seed treatment is not practiced, and, in the areas subject to soil contamination, even with seed-borne smut eliminated, 20 to 30 per cent of smutted heads is not uncommon. Susceptible varieties artificially coated with their maximum load of spores may produce 90 to 99 per cent of smutted heads.

Grain from badly smutted fields is conspicuously blackened when threshed due to the smut scattered from broken smut balls and will also

contain some unbroken balls. Such smutted grain is of less value for milling purposes, of less value for feed, if conspicuously smutted and, consequently, when placed on the market, suffers a dockage in price in accordance with the degree of smutting. Explosions of threshing machines followed by fires may result during the threshing of smutty wheat, especially in the arid regions, owing to the formation of an explosive mixture of dust and air which is ignited by static electricity. The separators may be burned, and the fire spread to the straw pile, the sacks of threshed grain or even to unthreshed grain in the field.

Etiology.—Bunt of wheat is due to either *Tilletia tritici* (Bjerk.) Wint., the rough-spored smut, or to *T. levis* Kühn (*foetans*), the smooth-spored smut, two very closely related species of the Tilletiaceae having nearly an identical life history. Another species, *T. indica* Mitra, has been reported on wheat in the Punjab, India, with spores double the size of the other species. The spores of *T. tritici* are spherical or nearly so, occasionally oblong to pear shaped, rough with a surface network of ridges and are 15 to 20 μ (maximum 22) in diameter. The spores of *T. levis* are smooth, globose to elliptic, occasionally somewhat angular, more variable in form and size, and measure 16 to 25 by 16 to 17 μ.

Under normal field conditions, infections with bunt occur only during the young or seedling stage from spores that were lodged on the surface of the seed or that were present in the soil at the time of seeding, but, even with the presence of this inoculum, favorable temperature and moisture conditions must be offered or the disease will not appear. Artificial infections have been made on growing plants by applying the inoculum to culms broken off near the base, by hypodermic injections and by inserting the inoculum inside the leaf sheath.

The smut spore germinates by the production of a hyphalike growth, the *promycelium*, which produces at its free end a fascicle of 4 to 12 long, narrow, curved spores or *sporidia* frequently united into H-shaped pairs. Infection threads may arise from these sporidia, or secondary sickle-shaped sporidia which are forcibly detached may be formed and later develop infection threads (see Fig. 100). Under favorable conditions, one or more infection threads (there is evidence of multiple infection) penetrate the young seedling and reach the growing point of the shoot. Here the fungous threads or hyphae keep pace with the growth of the host but give little or no external evidence of their presence until the production of heads, when they enter the ovaries and begin the development of the spores which reach maturity at or slightly before harvest-time. The smut fungus is heterothallic, no infection resulting from the mycelium produced from a single sporidium but only by mixed cultures or cultures resulting from the pairing of plus and minus strains. The two species and also the physiological strains of each may be hybridized.

During threshing, some of the smut "balls" or "berries" will break, and the resulting smut dust or spores (6 to 9 million from a single ball) may either be carried out through the stacker or lodged upon the surface of normal grains, collecting especially in the groove or suture and the terminal brush or tuft of hairs. The numbers may be too few to be visible to the naked eye (5000 or less) or sufficiently abundant to blacken

Fig. 100.—Various stages in the germination of spores of stinking smut (*Tilletia tritici*). *a*, spore surface showing characteristic reticulate ridges; *b*, *c*, stages in the formation of the promycelium; *d*, mature promycelium with terminal fascicle (*s*) of H-shaped sporidia; *e*, a separated sporidium which has germinated to form secondary sporidia (*ss*); *f*, a separated sporidium which has given rise to an infection thread (*in*); several secondary sporidia which have started to germinate and have produced infection threads.

the seed (up to 250,000 or more per seed). Wheat from smutted fields may also contain unbroken smut balls or sometimes fragments, while even wheat from smut-free fields may be carrying spores blown in from other smutty fields. Such contaminated seed is always a potential source of smut if used for seed.

When smut-free seed is used there are two possible sources of infection: (1) *residual smut* or spores from a previous crop on the same ground; and (2) *wind-blown* spores from either local or distant threshing opera-

tions. Spores in heads or unbroken balls in the soil may survive a year or more, but free smut spores in the moist soil will lose their infective properties after fifty to sixty days, and will not survive ordinary winter temperatures. Wind-blown smut is the most important source of soil contamination, especially in regions in which summer fallow is practiced, notably the Palouse country, in which the fall of smut during the threshing period may amount to over 5 million spores to each square foot of exposed surface. In such fields there is a possibility of infection even though carefully treated seed is used.

Predisposing Factors.—The amount of smut produced by a given seeding will depend upon the spore load, or the number of viable spores on each grain and also on the extent to which viable spores may be present in the soil. The actual percentage of infection is influenced by: (1) the amount of soil moisture during the germination period, the least smut with a very dry soil, the maximum amount with moderate moisture, but less again in a very wet soil; and (2) the temperatures which prevail during germination and emergence and, consequently, by the time of seeding. The optimum temperature for germination of wheat is 25°C. and for smut 16 to 20°C., therefore, if wheat germinates at its optimum or above, it will be likely to escape by passing the susceptible stage before the smut has reached the infection state.

Some regions are exceptionally free from smut (eastern center and south of India; southern Russia; and the Spokane Valley in Washington) and it has been shown that this is due to high soil temperatures which generally prevail at the time of seeding. The graphs (Fig. 101) show the relation of time of seeding to the per cent of smut in the crop using carefully treated Hybrid 143 at Pullman, Wash.

The low per cent of smut in the early seeding was due to two factors: (1) the relatively high soil temperatures; (2) the fact that the spore fall in August had not seriously contaminated the summer fallow. The reduction of smut during the late seedings is explained by the gradual loss of infective power of the smut spores and the unfavorable temperature conditions.

Physiologic Races.—The definite recognition of physiologic races of bunt is a comparatively recent development. The first evidence of physiological specialization in bunt was published by Faris in 1924. Since that time, this phase of the bunt problem has been studied rather extensively by workers in America, Germany and France. In Washington alone, 10 races of *Tilletia tritici* and 6 of *T. levis* have been recognized by the use of differential hosts. Because of lack of a standard system of identifying physiologic races, it is uncertain how many of the races described elsewhere may be duplicates. A recent comparative study recognized 11 races of *T. tritici* and 9 of *T. levis* (Rodenhiser and Holton, 1937), but this has been expanded by further study to 14 of the former and 10 of

the latter (Holton and Rodenhiser, 1942). It is certain that physiologic races are numerous, and experimental evidence indicates that the number is increasing. The appearance of new physiologic races, or their introduction from some other locality, is the explanation for the high percentage of smut in varieties formerly thought to be resistant. The numerous physiologic races certainly complicate breeding for resistance.

Fig. 101.—Graphs showing the percentage of bunt in periodic plantings for three different years.

It has recently been shown that other features than pathogenicity or host-tester reaction may serve to separate races. The additional differentiating characters are spore-ball characteristics, spore-wall reticulations, size of the spores, length of the promycelia, nuclear behavior in the promycelia, cultural characters and the relative stunting of the host affecting both height and tillering (Holton, 1935).

Host Relations.—The various species of Triticum vary from complete freedom from smut (possible immunity) to nearly 100 per cent of infection. Susceptibility is found within all the following: common wheat (*Triticum vulgare*), club or square-head wheat (*T. campactum*), poulard or English wheat (*T. turgidum*), durum wheat (*T. durum*), Polish wheat (*T. polonicum*), einkorn (*T. monococcum*), spelt (*T. spelta*) and emmer (*T. dicoccum*).

Bunt has also been reported on winter rye, rye-wheat hybrids, and on species of Aegilops, Agropyron, Hordeum, Lolium and Sitanion. The "compactum" types are generally very susceptible, the "vulgare" types variable, while durum wheat has been reported as resistant, although susceptible to some races.

Resistance is not correlated with quick germination or rapid growth, nor is it correlated with anatomical characters, but rather with chemical peculiarities of the host. Many reports on varietal susceptibility have been published, but these are largely invalidated by our recent discoveries of physiologic forms. Some varieties are susceptible to all known physiological strains of bunt, for example, Hybrid 128, while other varieties are immune to some and susceptible to others, for example, Albit and Ridit. Resistance to a strain of smut does not appear to be constant, but it has been lessened by successive passage of the strain through the same host.

Three types of resistance have been recognized: (1) resistant when fall-seeded but immune where spring-sown (Turkey); (2) resistance the same in either fall or spring; and (3) susceptible when fall-seeded, but highly resistant when spring-seeded (Hope, Martin).

Seed of the same varieties from different sources, when tested side by side and given equal opportunity to develop smut, may show great variation in the percentage of infection. For example, seven strains of Turkey showed 14.28 to 64.92, while six strains of Marquis varied from 18.39 to 42.55 per cent of smut. Susceptibility is equalized somewhat by growing the varieties a second or third year in the same environment.

Prevention or Control.—Use should be made of the several control practices in accordance with their adaptability to different environments. The following should be given consideration:

1. *Crop Rotation.*—Wheat following wheat may be smutty from smut produced by the previous crop, even with an intervening summer fallow. Rotation with some other crop is desirable.

2. *The Use of Clean Seed.*—Wheat that is visibly smutted, that is, shows the smut on the surface of the normal grains by naked-eye test should be rejected for seed if visibly clean or smut-free seed can be obtained. Even visibly clean seed may be carrying as many as 5000 smut spores per grain and might produce 10 to 15 per cent smut if seeded without treatment. If wheat showing unbroken smut balls must be used for seed, the smut balls can be removed by a fanning mill cleaner, by proper adjustment of air velocity, screen size and speed.

3. *Seed Disinfection.*—Treatment with some standard fungicide should be practiced unless the spore load is known to be below the danger point, or seeding is carried out under conditions which will prevent infection. Acceptable fungicides should give nearly perfect protection

except where there is a soil contamination. The following are the most important fungicides which have given effective control of seed-borne smut in farm practice:

a. Steeps or liquid treatments

(1) Copper sulphate or bluestone, 1 pound to 5 or 10 gallons of water for 5 to 10 minutes, with maximum time for the weaker strength.

(2) Bordeaux, 4–4–50 to 8–8–50 formula, for 10 to 15 minutes.

(3) Formaldehyde, 1 pint or pound to 40 gallons of water (1–320) for 10 minutes.

(4) Uspulun or germisan, 0.25 per cent solution for 20 minutes.

b. Dust disinfectants

(1) Copper carbonate, 50 per cent to 20 per cent copper, 2 to 3 ounces per bushel.

(2) Basic copper sulphate (Basul), 2 to 3 ounces per bushel.

(3) Copper oxychloride, 0.2 per cent by weight of grain treated.

(4) Organic mercury dusts (*e.g.*, ceresan 2 to 3 ounces per bushel, new improved ceresan, ½ ounce per bushel). The use of mercury dusts is forbidden in France because of their toxicity to man.

Hundreds of other chemicals have been tested and used to a limited extent in various countries.

For the liquid treatments, open-tank or sack methods are much better than sprinkling. Many types of treating machines are available for both liquid and dust disinfection. Either cooperative or commercial seed disinfection is provided in certain sections in the United States and in several European countries.

Much seed injury may result from the use of either bluestone or formaldehyde and a protective afterbath must be employed or the rate of seeding increased. Copper carbonate and basic copper sulphate dusts cause no seed injury and have the following additional advantages: (a) the elimination of the inconvenient and disagreeable soaking methods; (b) the convenience of being able to treat seed days or even months before it is to be used; (c) the elimination of danger of seeding in the dust; and (d) an improved germination over untreated seed. These advantages hold also for the new improved Ceresan, except that seed treated with this fungicide must be seeded soon after dusting.

4. *Cultural Practices.*—The following may reduce the amount of smut: (a) seeding in relatively dry, rather than in moderately moist, soil; (b) shallow rather than deep seeding; (c) early seeding when the ground is warm or before the smut shower; (d) replowing of summer fallow before seeding; (e) trench seeding rather than surface seeding; (f) a fall stubble crop rather than summer fallow; and (g) late-fall rather than mid-fall seeding.

5. *Selection of Resistant Varieties and Breeding for Smut Resistance.*— First consideration in any environment must be given to varieties which will show high production. The testing of varieties is showing that high production can be combined with resistance to a large number of the physiological races of bunt. The ultimate aim of the plant breeder is the production of immune varieties of high producing power and prime quality, but it seems doubtful whether complete immunity will ever be attained. At present the experience of the local experiment stations should guide the growers in the selection of varieties best suited to their environment.

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masses. Partially smutted heads are sometimes found and smut masses are occasionally found on leaf blades, leaf sheaths or culms. Even one month after seeding, infected plants can be detected by (1) absence of awns in awned varieties; (2) brown streaks extending for the whole length of the ear; and (3) the flattened form of the spikelets (Saburova, 1937).

The smut masses have reached maturity and the spores are being scattered by the wind by the time normal heads are in flower, and by harvesttime the smutted heads are inconspicuous owing to the weathering away of the smut masses. The amount of smut varies under field conditions from a trace to as high as 15 to 20 per cent, depending upon the location and the variety, but never equals the high losses from bunt.

Loose smut causes an increased transpiration over normal plants and a greatly reduced weight by harvesttime; also increased winterkilling has been reported. The loss from loose smut must be based on the reduction in yield, since quality is not affected as in bunt, unless one considers the effect on value for seed purposes. It is customary to figure the reduction in yield equivalent to the per cent of smutted heads, but, in low degrees of smutting, the loss would be much less.

Etiology.—Loose smut of wheat is caused by *Ustilago tritici* (Pers.) Jens., a fungus which infects the young ovary at the time of flowering and develops its mycelium within the seed, thus producing an intraseminal infection. The smut spores are blown about by the wind just at the time when the normal heads of adjacent plants are in the flowering stage and some of the innumerable spores will lodge between the glumes or chaff and reach the feathery stigmas where they germinate and send an infection thread down the tissue of the style into the ovule. Here the smut fungus continues to grow within the young embryo plants, but it becomes dormant when the seed is matured. Seeds carrying an intraseminal mycelium appear perfectly normal, but, when planted the next season, the dormant mycelium resumes activity and keeps pace with the growth of the young seedling. The mycelium is present in all organs of the embryo, especially the growing point, and, with the organization of the heads, makes a vigorous development and destroys the various parts as already noted. The spores of the dusty smut masses or sori are globular or oval, pale olive-brown, single-celled, 5 to 9 μ in their greatest diameter, are marked with minute spines and appear lighter on one side.

The spores germinate freely in water or nutrient solutions as soon as mature, but they do not retain their viability for more than five or six months, even suffering a marked loss of viability during this period. Germination takes place by the formation of an infection thread which is pushed out from the clear side of the spore or from one of several clear spots in the spore wall. The germ tubes from spores lodged on the feathery stigmas grow down the style to the ovary, penetrate the integu-

ments of the ovules, and thence, through the endosperm, nucellus and scutellum, penetrate the embryo. In the matured seed all parts of the embryo except the root contain hypae, and there is a copious mycelium in the scutellum.

The time of seeding of spring wheat does not seem to have any effect on the per cent of smut which appears in the crop, except in the case of late fall seeding of spring varieties, when no smut develops according to

FIG. 102.—*Ustilago tritici*, loose smut of wheat.

some Russian experiences. Some tests indicate that time of seeding in fall grain influences the development of the parasite, the lowest per cent of smut appearing in the late seedings. This can be explained by supposing that the low temperatures of late fall, which are sufficient to ensure germination of the seed, are not sufficiently high to start the growth of the intraseminal mycelium, with the result that the growing point "runs away from the pathogen" and the pathogen is never able to catch up with it even though favorable temperatures may later start it into activity.

The severity of loose smut is influenced by moisture and temperature. factors during the germinating period of the wheat and during the

period of spore dissemination. Lack of rain or dews during the period of spore dissemination are probably responsible for the light infections in much of the plains country or semiarid west. Some tests have shown less smut in light clay-sand soil than in heavy soil and a reduction by the use of a complete fertilizer. Some recent studies have shown that the spores may be wind-borne from infected fields to adjacent smut-free

Fig. 103.—Spores of *Ustilago tritici* at rest and after various stages of germination in water. *a*, group of spores showing various shapes and sizes; *b–r*, early and later stages of germination. (*After Stakman, Minn. Agr. Exp. Sta. Bul.* **133**.)

plantings and induce new infections at distances of 100 meters or more, resulting in uniform infection from strong winds, and spotted infection from light winds (Oort, 1940).

Biological Strains.—Recent cultural studies have led to the conclusion that loose smut of wheat and loose smut of barley are not distinct species but physiological forms of the same morphological species. On the basis of cultural studies in Minnesota three strains from wheat and six from barley have been recognized. More recent German work based on pathogenicity on a series of differential host varieties, recognized three physiological strains from Germany and a fourth one from Turkey.

Later studies have shown still more physiological strains which add a complicating factor to breeding for resistance. By crosses between resistant and susceptible varieties, German workers have been able to develop a spring wheat resistant to strain 1 and 2 of the loose smut of wheat.

Varietal Resistance.—Field experience in different areas has shown considerable variation in the percentage of loose smut in different varieties. Some claims have been made that immunity in loose smuts is due to closed flowers, which prevent the smut spores from reaching the stigmas during the susceptible period, but later work pointed out that this does not hold for wheat, as no varieties have closed flowers. This structural explanation may hold, however, for some varieties of barley. A number of reports have been made on varietal resistance, but most of these have been based on single physiological strains, hence are not generally applicable. Very susceptible, moderately susceptible and immune varieties have been recognized. Ridit and several German varieties, Strubes' Silesian, Wohltmann's Green, Hungarian Theiss and Grüne Dame have been reported as immune, while Pentad, a durum wheat, Hussar, Black Persian and Peragis are very resistant. There is no correlation between the hydrogen-ion values of the cell sap of susceptible, resistant or immune varieties. Resistance is recessive and inherited according to the Mendelian ratio.

Prevention or Control.—Since the fungus is carried in the seed in the form of a dormant mycelium, the disinfection methods used for smuts with surface-borne spores are not effective. An effective method must kill the internal mycelium without killing the embryo. This may be accomplished by the *modified hot-water* treatment (Jensen method) or hot-water baths plus certain chemical adjuvants.

The modified hot-water treatment may be carried out as follows: (1) soak the wheat in water at 68 to 86°F. for 4 to 6 hours; (2) immerse in warming vat for 1 minute at a temperature 5 to 10° under that of the treating vat; (3) immerse in treating vat for 10 minutes at 129.2°F. or within a range of 124 to 130°F.; (4) drain and dip at once in cold water or spread out in a thin layer to cool and dry, preferably the former as it gives less seed injury. Plant as soon as the seed will run freely through the drill, or dry thoroughly and store for later use. The exact method of providing the hot water and handling the grain can be varied. This hot-water treatment will cause more or less seed injury depending on variety, threshing injury, care in treating, etc., and may be expected to cause a reduction in germination of as little as 10 per cent in the best possible adjustments or of 40 per cent in the average standard procedure.

A single bath, hot-water or steam treatment has been found effective and lessens seed injury. These two methods are suitable only for coop-

erative plants handling large quantities of seed: (1) hot water for 110 minutes at 118.4°F. or 95 minutes at 120.2°F.; or (2) exposure to a satu · rated recirculating atmosphere of steam for 1 to 4 hours at 46°C., 1 to 2 hours at 47°C. or ½ hour at 48°C. Some more recent trials in Germany have shown that the effectiveness of the hot-water treatment may be increased by the addition of 2 per cent isopropyl alcohol or 3 per cent methylated spirits with the treatment time ranging from ¼ to 4 hours according to the temperature used. Good results were also obtained by heating in closed containers with 5 to 10 liters of water per 100 pounds of seed at 45°C., but, with addition of alcohol, the smut was completely eliminated. In some of our eastern states and in Germany the hot-water treatments have been carried out in community plants making use of creameries, canneries, mills or other establishments furnishing live steam, or by the construction of special rotary machines. Some recent reports from India have recommended four methods of seed treatment including two modifications of the hot-water treatment and two methods of sun heating.

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COMMON SMUT OF CORN

Ustilago zeae (Beckm.) Ung.

Three smuts affect corn: (1) the common or boil smut; (2) the head smut, *Sorosporium reilianum* (Kühn) McAlp., also on sorghums; and (3) kernel smut (*Ustilago fischeri* Pass.) known mainly from Italy and the West Indies.

Fig. 104.—*Ustilago zeae* on an ear of corn.

Corn smut undoubtedly originated in America upon native stock but was first described by French botanists as early as 1754. The earliest American record was by Schweinitz in 1822. During early years, it was believed to be caused by a physiological disturbance (up to 1832); later its parasitic nature was recognized, and it was thought to be seed-borne; while in more modern times (Brefeld and later) the purely local nature

Fig. 105.—*Ustilago zeae.* *A*, on corn tassel; *B*, on midrib of a leaf blade.

of the infections was demonstrated and many important contributions made by European and American workers.

The disease is prevalent in nearly all countries to a greater or lesser extent wherever corn is grown, but, in 1910, it had not yet reached Australia.

Symptoms and Effects.—The disease appears on various aerial parts of the corn plant as either small or large tumors, at first whitish owing to the covering membranes, but later darker and then black from the development of the enclosed smut mass. Recently the symptoms have been classed as *exposed* and *concealed*, the former including irregular yellowish or reddish stripes or blotches, brownish lesions and galls, the latter including small nodal galls and minute pustules of chlamydospores in the leaves of axillary buds (Davis, 1936). The tumors may occur on leaves, at nodal buds on the stem, on the tassel or male inflorescence and on the various parts of the ear or pistillate inflorescence, and they vary in size from minute pustules on the leaves to others on the stalks or ears as large as a child's head. When the tumors reach maturity the covering membrane dries and breaks, exposing the dry, powdery mass of spores.

The impression frequently prevails among farmers that only ears and tassels are smutted, but this is due in large part to the conspicuous character of the tumors on these structures.

Loss from Corn Smut.—The disease may be present in traces only or, under very favorable conditions with continuous cropping to corn, as high as 60 to 70 per cent of the ears may be affected. Losses of 5 to 15 per cent have been recorded in some sections of the corn belt, while the reduction in yield caused by smut for the entire United States from 1918 to 1924 was estimated at 2.1 per cent. The injury manifests itself both directly and indirectly and will vary according to the number and location of the infections. Large boils above the ear are more injurious than when located below the ear. Smut may be the cause of premature killing in severe infections and may also cause sterility, decrease the normal luster of kernels and render the ears more susceptible to rots.

Etiology.—The causal fungus, *Ustilago zeae* (Beckm.) Ung., causes localized infections on the affected structures and is not systemic as in the bunt of wheat.

A mature smut tumor consists of a thin covering membrane of host origin of thin-walled cells enclosing the mass of powdery pores mingled with the remains of collapsed host parenchyma and vascular elements. The spores are set free by the rupture of the covering membrane and may be mingled with the soil, scattered by the wind, carried to the feed lot with smutty fodder and there mingle with the barnyard manure, which later may be spread upon the field.

The spores, which are brown, spherical to ellipsoid, 7 to 12 μ in diameter and covered with small spines, germinate either at once or after a period of dormancy. A typical *promycelium* may be formed consisting of four somewhat elongated cells from which both terminal and lateral secondary spores or *sporidia* are budded as somewhat fusiform bodies of variable size (Fig. 106). In some cases a promycelium may be formed with one or more hyphal branches in the place of sporidia; in others the chlamydospore may form two opposite promycelia; and in some the chlamydospore may function as the basal promycelial cell (Kernkamp and Petty, 1941). Under favorable conditions of nourishment, the promycelium may branch and form an abundance of sporidia. These may form infection threads at once, or, if supplied with a nutrient solution, they will bud profusely, in a yeastlike fashion, to form secondary sporidia which may behave like those produced directly from the promycelium.

Both the primary sporidia and the secondary sporidia may be scattered by the wind, and, whenever they reach the susceptible growing tissue of a corn plant, they may germinate and produce localized tumors, either by penetration through stomata or through wounds. The period between infection and the formation of mature sori varies from seven

days to three weeks, depending upon environmental conditions. Following the penetration of the epidermis by the germ tube the parenchyma cells are stimulated to active cell division and the smut tumor is formed consisting of mingled hyphae and host cells. After the mycelium has permeated the tumor tissue, spore formation is initiated. The compact, branched hyphae become segmented into short, uninucleate cells, adjacent pairs becoming merged by the solution of the separating wall into irregular, cylindrical, binucleate cells. Gelatinization of the walls occurs and each binucleate mass surrounds itself with a spore wall,

Fig. 106.—Germination of spores of *Ustilago zeae*. *a–f*, germination in manure decoction at 19 hours; *g*, formation of germ tube in water at two days; *h* and *i*, promycelia at seven days producing sporidia; *j* and *k*, typical sporidia, the three lower in *k* germinating. (*After Stakman, Minn. Agr. Exp. Sta. Bul.* **133**.)

the gelatinous remnants are used up, the two nuclei unite and the isolated spores first constitute a pasty mass which later becomes powdery in the mature tumor.

Smut spores will germinate if taken at once from new tumors but will retain their viability and infectiveness in the smut masses for several years (five to seven). The optimum temperatures for germination lie between 20 and 34°C., according to region of origin or strain, the maximum between 36 and 38°C., while the minimum is about 8°C. This relatively high optimum probably explains why corn smut is more severe in the warm corn-growing areas than in the cooler regions.

Few if any smut spores remain viable after passing through the digestive canals of horses or cattle, but they will germinate readily in the compost heap, and the sporidia will continue to bud under these conditions but do not withstand the high temperatures of fermenting

farmyard manure.   In this way enormous number of spores produced by a tumor may be greatly increased and a more widespread dissemination brought about if the contaminated fertilizer is spread upon the field. Under natural field conditions, infections do not take place until plants are 1 to 3 feet high, but new infections may develop any time up to the tasseling stage.   During the susceptible period, infection may take place at any point where there is young growing tissue, but matured tissues cannot be penetrated.

Host Relations and Conditions Favoring Smut.—Teosinte (*Euchlaena mexicana*) is the only other host attacked by common corn smut.   Differences in susceptibility of varieties of corn have been noted, some being nearly immune, and it is generally agreed that flint varieties are more susceptible than dent varieties.   It has been reported as especially severe on varieties from Mexico, South America and the Philippines.

Corn smut is increased in severity or favored by: (1) vigorous growth with increased succulence; (2) early planting or a prolonged growing season; (3) close planting; (4) thin husks that split easily or are too short to cover the ear tip; (5) mechanical injuries, by hail, for example; (6) strong winds; (7) high relative humidity; and (8) irrigation as contrasted to nonirrigation.   Resistance to smut may be caused by reduced vigor, from any cause, and selfed lines may be smut resistant because of this lack of vigor and such strains may give lower yields.   That resistance is in part physiological has been demonstrated by the inhibiting action of the filtered juices of the vegetative parts of resistant varieties upon the growth of the smut fungus in cultures.

Physiologic Strains and Mutation.—The corn-smut fungus is generally heterothallic; that is, monosporidial lines are generally incapable of producing infection with the production of smut boils, but may produce flecking.   A few monosporidial lines have shown parasitism by artificial inoculation.   The use of plus and minus monosporidial cultures, or those of opposite sex, gives successful infection.

Corn smut has been shown to include many physiological strains. The first reports from Minnesota workers recognized 15 forms indicated by cultural characters and at least 7 forms by their varying parasitic behavior.   In cultures, sectors and patch mutants arise in most monosporidial lines, and these appear to be true mutants.   It is the conclusion that the corn-smut fungus now comprises many lines and that new ones are continually arising by hybridization and mutation.

Corn Smut Not Poisonous to Cattle.—An erroneous belief has been prevalent that corn smut is poisonous to cattle.   Many feeding experiments in the corn-producing states have shown that it is only rarely injurious.   It contains a small amount of an active principle resembling ergot in its action but not in sufficient quantity to be of danger to cattle.

Extracts of smut have produced toxic effects when fed to laboratory animals, except white rats. Studies in Yugoslavia have reported a diseased condition in children known as ustilaginism.

Control.—Since seedling infection from seed-borne spores does not occur, seed disinfection can be of no value. Infections are local and on aerial parts and experiments have shown that corn can be protected by spraying with Bordeaux, but this is not practical in farm plantings. The following control practices are recommended: (1) crop rotation or corn planted not oftener than once in three or more years on the same land; (2) frequent inspections and the collection and destruction of smutted ears and stalks (by burning) before spore dissemination; (3) avoidance of smut-contaminated fertilizers for corn ground; (4) use of planting and cultural practices that are the least favorable to infection; and (5) the selection of resistant varieties.

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ONION SMUT

Urocystis cepulae Frost

Onion smut is caused by one of the few important smut fungi that attack other than cereal hosts. The disease first attracted attention as early as 1857 in Massachusetts and has spread to practically all the onion-growing regions of the northern United States as far west as Washington and Oregon. It is not known in the southern states and

has never been found in California. It has been reported from England, continental Europe, Australia, New Zealand and Japan, but has been less serious in these countries than in America. In addition to the common onion, the disease is known to attack other cultivated species of Allium, including chives, leeks, shallots and Welsh onions in addition to various wild species.

Fig. 107.—Symptoms of smut on mature onions. *A*, showing distorted leaves with ruptured pustules exposing black spore powder; *B*, smut pustules on bulb; *C*, healthy onion for comparison. Note failure of diseased plants to develop normal bulbs. (*After Anderson and Osmun.*)

Symptoms and Effects.—The first evidence of the disease is generally noted on the seed leaf (cotyledon), which may show a slight distortion and swelling. An affected seed leaf when held to the light will show one or more black, opaque, elongated, internal areas. These affected plants may "damp off," but if not the smut alone may cause them to shrivel and die in many cases, thus causing thin stands. If one of these blackened seed leaves is crushed, it is found to be filled with a black powder (spores).

In less severe initial attacks, the plants may not be killed in the cotyledon stage and growth continues, with the development of smut

in the succeeding leaves. Such affected plants may be stunted and form only short distorted leaves, and very poorly developed bulbs, which are not harvested because of small size or invasion by rot. In some plants the effect may be less pronounced, and as the plants increase in size, the black smut lesions may be several inches long or even occupy almost the full length of the leaf. They may also involve the bulb proper and appear as gray, raised streaks on the lighter scales. With the death of the leaves and the rupture of the scales, the black spore powder may fall out and be mingled with the soil.

Etiology.—Smut of the onion is caused by one of the true smut fungi, *Urocystis cepulae* Frost, which infects the host only in the seedling stage. The onion smut was transferred to the genus Tuburcinia as *T. cepulae* (Fr.) Liro in 1922, and this name has been used by European writers, although not adopted by American workers. ✓The smut dust from the infected host parts consists of numerous compound spores or spore balls (chlamydospores) each consisting of a central, dark brown, fertile cell, surrounded by numerous smaller, transparent or slightly brown sterile or accessory cells. The spores when set free into the soil may germinate at once or may remain viable and germinate months or even years later.

Two types of germination have been recorded: the *first* by relatively young spores; the *second* by spores that were several years old. In the *first* type a globose hyaline body is protruded from the central fertile cell, and then gives rise to one to eight germ tubes which become branched and septate, but produce no sporidia. In the *second* type, the large globose body was not produced but only a single hypha which finally became septate and branched after considerable growth in length and ultimately developed a mycelium the same as in the first type. The formation of sporidia as described by some earlier workers has not been substantiated by the later studies. Infection takes place by the hyphae penetrating the tender epidermis of very young seedlings, the period of susceptibility depending upon the temperature,. but ranging from 17 to 24 days after seeding according to certain tests. It should be pointed out, however, that the period of infection covers the time from the beginning of germination of the seed to the stage when the first leaf has emerged from the side of the cotyledon.

The mycelium which has penetrated the epidermis of the cotyledon ramifies in all directions and finally forms dense knots of fungous cells which are transformed into the chlamydospores as previously recorded. The mycelium from a single infection does not spread for more than a quarter inch; hence longer lesions represent the fusion of adjacent infection centers. If the mycelium does not reach the growing zone, the infected cotyledon tissue may be sluffed off and normal growth may continue. If, however, the mycelium reaches the growing zone, each

developing leaf will carry some of the mycelium which will continue to spread and finally sporulate.

FIG. 108.—Germination of spores.   (*After Anderson.*)

The temperature that prevails during the infection period influences the amount of infection.   Some infection may result at temperatures of 10 to 12°C. or nearly as low as the beginning of seedling growth; hence there is no low point that will entirely eliminate infection.   Early seeding under certain conditions (New York) has been shown to greatly reduce infection, since onion seed will germinate at 8°C. or above, while smut germination and infection require a slightly higher temperature.

"The optima for chlamydospore germination, hyphal fragment germination, and vegetative growth of the thallus lie between 13 and 22°C. Above 25°C. there is a decided reduction in the amount of germination which occurs, and the growth of the hyphae becomes more meager as the temperature rises." (Walker, 1926.) It is undoubtedly this temperature relation which has excluded the disease from the intensive, southern, onion-growing regions.

Onion smut may be spread by the spores which adhere to the feet of man or animals, or the spores may be spread by farm implements, surface drainage, wind-blown soil, and may also be carried on the seed and by infected sets. Smut-free sets will not be infected even if planted in contaminated soil. The onion-smut fungus will live and grow for years in the soil that is rich in organic material and may originate from spores or as mycelium from the host, either of which may initiate infections. No conidia are produced, but detached or separated mycelial cells may serve to disseminate the fungus by the same agencies as already noted for the chlamydospores and continue its growth, and infections may result from this mycelium. The persistence of the smut in the soil may be due either (a) to chlamydospores which have remained viable; or (b) to the saprophytic mycelium and mycelial fragments.

Control.—Five different control measures may be listed: (1) The destruction of infected onion refuse (tops and discards) from harvesting operations or from warehouses by burning to prevent contamination of the soil. (2) The use of sets when this method is practical, since sets are not attacked even on contaminated soil. (3) The transplanting of seedlings grown in smut-free hotbeds or greenhouse to the field, a method common in the South, but used to some extent in northern sections. (4) The use of a chemical disinfectant by application to soil in the drill row. Many chemicals have been tried, the most promising results accruing from flowers of sulphur, lime-sulphur and formaldehyde, but the latter is the only one that has stood the test of time and is now generally recommended and extensively used. The formaldehyde is added to the drill row during seeding by the use of a formaldehyde tank from which the rate of flow can be controlled. A very efficient apparatus has been described and used by Anderson and Osmun (1924). "One gallon of formaldehyde is added to 50 gallons of water and applied at the rate of 1 barrel to 1 acre if the soil is very dry; or $1\frac{1}{4}$ barrels if the soil is only medium moist; or $1\frac{2}{3}$ barrels if the soil is wet and heavy. (5) Some promising results have been obtained by treating the seed with a number of different dust fungicides, but none have come into general use.

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IMPORTANT DISEASES DUE TO SMUT FUNGI

For key references to these diseases see F. D. Heald, "Manual of Plant Diseases," 2d ed., pp. 757–761.

1. Ustilaginaceae

Principal host	Common name of disease	Scientific name of causal organism
Oats.....................	Loose smut	*Ustilago avenae* (Pers.) Jens.
Oats.....................	Covered or kernel smut	*U. levis* (K. and S.) Mag.
Wheat and Rye............	Loose smut	*U. tritici* (Pers.) Rost.
Barley...................	Loose smut	*U. nuda* (Jens.) K. and S.
Barley...................	Loose smut	*U. nigra* Tapke
Barley...................	Covered smut	*U. hordei* (Pers.) K. and S.
Corn.....................	Common or boil smut	*U. zeae* (Peck) Ung.
Corn.....................	Kernel smut	*U. fischeri* Pass.
Timothy and other grasses...	Leaf smut	*U. striaeformis* (West.) Niess.
Millet...................	Millet smut	*U. crameri* Körn.
Sorghum..................	Kernel smut	*Sphacelotheca sorghi* (Lk.) Cl.
Sorghum..................	Loose kernel smut	*U. cruenta* (Kühn) Potter
Sorghum, corn............	Head smut	*Sorosporium reilianum* (Kühn) McAlp.

2. Tilletiaceae

Wheat...................	Bunt or stinking smut	*Tilletia tritici* (Bjerk.) Wint.
Wheat...................	Bunt or stinking smut	*T. levis* Kühn
Rice....................	Black smut	*T. horrida* Tak.
Wheat...................	Flag smut	*Urocystis tritici* Koern.
Rye.....................	Leaf or flag smut	*U. occulta* (Wallr.) Rab.
Onion...................	Onion smut	*U. cepulae* Frost
Spinach.................	White smut	*Entyloma ellisii* Halst.
Dahlia..................	White smut	*E. dahliae* Syd.
Sunflower...............	White smut	*Entyloma spp.*

Fig. 109.—Oat smuts. *A*, kernel smut (*Ustilago levis*); *B*, loose smut (*Ustilago avenai*

CHAPTER X

DISEASES DUE TO BASIDIOMYCETES

RUST FUNGI, UREDINALES

Because of affinities with the true basidium fungi, the rust fungi have sometimes been designated as Protobasidiomycetes.

Nature and Importance.—The common name of the order has been suggested by the conspicuous orange or reddish spore pustules or sori which are characteristic of certain stages. The true rust fungi are obligate parasites affecting a few ferns, but attacking in the main various species of seed plants (Gymnosperms and Angiosperms). The ravages of rust are known wherever plants are cultivated, species of the order being responsible for important diseases in nearly all groups of economic plants and in addition there is a wealth of forms which are confined to weeds or to plants of no importance.

General Characters.—The following are the important characters of the group: (1) an intercellular, branched, septate mycelium (more rarely intracellular) containing yellowish or orange-red oil drops; (2) polymorphism of spores, typical rusts producing a succession of five different forms in the course of the life cycle; (3) the germination of the teliospore to form a *promycelium*, or a sporulating stage independent of the host; and (4) the development in certain species of heteroecism, or the separation of the spore forms on two separate and unrelated hosts.

The Mycelium and Its Effects.—The internal, intercellular, septate mycelium is frequently brought into nutritive relations to the host cells by the formation of specialized sucking organs or *haustoria* which penetrate the cells. These haustoria may be globular, tubular, inflated, branched or in ball-like coils. Uninucleate and binucleate mycelial cells are characteristic of certain stages in the life cycle. The first effect of rust mycelium is not normally a killing of the host cells but, frequently, a stimulating effect which may cause abnormal growth and continuation of vegetative development. The mycelium may be purely local, or it may spread extensively throughout special organs, certain shoots or even the entire plant. No malformation may result or the host parts may be variously malformed, galls, hypertrophied stems or witches'-brooms appearing with the suppression or deforming of leaves or flower parts. In many such cases the mycelium may be perennial within the host tissues and continue to advance as long as the host remains alive,

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or, in other cases, it soon pervades the entire plant and may continue
to sporulate from season to season.

The Spore Forms.—A typical rust may develop five spore fruits
and spore forms in its life cycle, but, in certain species, one or more of
the forms may be omitted. The succession is shown in the following
tabulation:

Symbol	Spore fruits	Spores	Stage
0	Pycnia (Spermogonia)	Pycniospores (Spermatia)	Cluster cup
I	Aecia (Aecidia)	Aeciospores (Aecidiospores)	Cluster cup
II	Uredinia (Uredosori)	Urediniospores (Uredospores)	Red rust
III	Telia (Teleutosori)	Teliospores (Teleutospores)	Black rust
IV	Basidia (Promycelia)	Basidiospores (Sporidia) .	

The *pycnia* are minute flask-shaped or disklike, subcuticular or sub-
epidermal receptacles opening to the surface by a pore or ostiole, and
produce rounded, oval or elongated bodies, the *pycniospores*, which are
extruded through the ostiole with a sweetish secretion or nectar in which
they are embedded.

The *aecia* are globular, cup-shaped, tubular or irregular fungous fruits
which burst through the epidermis or periderm of the host. In the
typical form an aecium consists of a membrane of fungous cells, the
peridium, enclosing or surrounding the central fertile portion which
produces the one-celled *aeciospores* in chains.

Four types of aecia are recognized: (1) the *caeoma* type, with peridium
absent or represented by a surrounding circle of sterile hyphae; (2) the
aecidium, cup-shaped with the peridium toothed on its free edge; (3) the
roestelia type, with an elongated cylindrical peridium, split into segments;
and (4) the *peridermium* type, with the peridium irregularly split or
broken (confined to Conifers).

The *uredinia* are groups of spore-bearing hyphae crowded together to
form naked spore pustules or sori (red rust) which give rise to the single-
celled, elliptical, ovoidal or spheroidal spores, the *urediniospores*, in some
species mingled with sterile hyphae, or paraphyses. The spores are
binucleate with yellow or orange contents and a colorless or slightly
brownish wall marked with minute superficial spines, needles or warts and
provided with 1 to 10 equatorial pores. In some rusts the urediniospores
are modified to form thick-walled spores, or *amphispores*, which germinate
after a resting period.

The *telia* are dark brown or black spore pustules of various size
and form somewhat similar to the uredinia and form sessile or stalked
spores, the *teliospores*. These sori are subcuticular, subepidermal or, in

the epidermal cells, are powdery or gelatinous and may remain embedded or covered or expose the spores by rupture of host tissue. The teliospores are one- to many-celled (see Fig. 111) with thick, dark, variously sculptured walls; each cell is binucleate when young but uninucleate when mature, and it is provided with one to four germ pores. *Mesospores* are one-celled teliospores formed by species which normally produce two-celled teliospores.

Fig. 110.—Semidiagrammatic representation of spore fruits of a typical rust. *A*, an aecium with aeciospores; *B*, pycnium with pycniospores; *C*, uredinium with urediniospores; *D*, telium with teliospores. (*After Wettstein, Handbuch der Systematischen Botanik.*)

The *basidium* or *promycelium*, formed by the germination of a teliospore or one of its component cells, is a four-celled, hyphalike structure, each cell of which produces a slender distal outgrowth, or sterigma, bearing an ovate, globular or kidney-shaped spore or *sporidium* (basidiospore). The sporidia are forcibly detached from their sterigmata and are wind disseminated, but are mostly short-lived.

In many rust species, if not in all, plus and minus pycnia are produced, and the transfer of the sporidia-containing nectar from plus to minus groups or vice versa is followed by the formation of aecia. Aeciospores germinate by an infection thread, and the new mycelium gives rise to either uredinia or telia. The urediniospores germinate at once to form

an infection hypha which enters the host, new uredinia soon appear, and the process may be repeated throughout the growing season. The teliospores germinate in some species in the fall as soon as mature or, more frequently, only after a period of winter rest, and they develop the promycelia and sporidia, which may start the life cycle again. All of the spore forms may be developed in sequence on a single host (autoecious types) or the cluster-cup stage, pycnia and aecia, may be on one host and the balance of the life cycle on a second, unrelated host (heteroecious types).

Fig. 111.—Types of teliospores. *A*, sessile or nonpedicellate forms; *B*, stalked or pedicellate forms. (*Adapted from various sources.*)

Heteroecism.—It is generally agreed that autoecious types are primitive and that later the spore stages become separated on two distinct hosts. This separation, or heteroecism, finds a more complete expression in the rusts than in any other forms of plant life, and it is estimated that over one-twelfth of all species of rusts are heteroecious. Under natural conditions the alternate hosts are members of the same plant society, but in certain cases they are brought together by agricultural or horticultural practices. Monocots bearing uredinia and telia usually have the pycnia and aecia on some dicot host, while many dicot hosts with uredinia and telia may have the aecial stage on certain conifers, although the reverse may be true. The following are notable heteroecious rusts: orange rust

of aster and goldenrod; blister rust of five-needle pines; poplar rusts; witches'-broom of fir and spruce; stem rust of wheat and other cereals; rust of apple; pea rust; rust of stone fruits; corn rust; and rust of beet and spinach. In some cases the aecial stage is the one important on economic hosts, while in others the uredinial or telial stages are of more importance.

Life Cycle Combinations of Spore Forms.—Two groups may be recognized: (1) *short-cycle species*, in which the pycnia are followed at once by telia or the pycnia may even be omitted; and (2) *long-cycle species* in which either aecia or uredinia or both are introduced between pycnia and telia. The grouping of the spore stages and the terminology of the spore combinations are presented in the accompanying tabulation.

Eu-forms		Opsis-forms		Brachy-forms	Hemi-forms	Lepto- and micro-forms	
Auto	Hetero	Auto	Hetero	Auto	Auto	Auto	Auto
0	0	0	0	0		0	
I	I	I	I				
II	II			II	II		
III	III	III	III	III	III	III	III
IV	IV	IV	IV	IV	IV	IV	IV
Long-cycle forms						Short-cycle forms	

Classification.—There is but little uniformity of practice concerning the grouping of the rust fungi into families, the same author making changes in successive publications. The following grouping is convenient and affords a partial expression of relationships:

1. *Endophyllaceae.*—No separate aecia and telia, but aecioteliospores germinating by a typical promycelium and sporidia. Genera: *Endophyllum* and *Kunkelia*.

2. *Coleosporiaceae.*—Teliospores germinate *in situ* by elongation and division to form four cells, each of which produces an elongated sterigma bearing a sporidium. Genera: *Coleosporium* and *Gallowaya*.

3. *Cronartiaceae.*—Teliospores unicellular, not pedicellate, produced in chains which remain separate or are united into lens-shaped, wartlike or columnar telia. Genera: *Chrysomyxa* and *Cronartium*.

4. *Melampsoraceae.*—Teliospores sessile, unicellular or two to four celled, produced singly or forming extensive subcuticular, intraepidermal or subepidermal layers one to several cells thick. Important genera: *Necium, Melampsora, Melampsorella, Phakospora, Pucciniastrum* and *Calyptospora*.

5. *Pucciniaceae.*—Teliospores stalked, one to several celled, borne singly on a pedicel or in groups on either simple or compound pedicels, subepidermal and covered or free, and erumpent or embedded in a gelatinous matrix. Important genera: *Uromyces, Hemileia, Puccinia, Tranzschelia, Gymnoconia, Gymnosporangium, Kuhneola* and *Phragmidium*.

6. *Uredinales Imperfecti.*—Teliospores unknown. A temporary grouping of spore forms which cannot be assigned to recognized families and genera. Form genera: *Caeoma, Peridermium, Aecidium, Roestelia* and *Uredo*.

In the most recent classification of Arthur all rusts included in families 1, 2 and 3 are merged with the Melampsoraceae and the Pucciniaceae.

Biological or Physiological Specialization.—The development of physiological strains within certain morphological species is even more pronounced in the rusts than in the powdery mildews. Rusts may be monivorous, that is, they may be confined to a single host species or even variety, while many are plurivorous, that is, capable of infecting many different hosts which may be related or unrelated. Plurivorous species may be autoecious, as in *Puccinia malvacearum*, or in heteroecious species the aecia may be developed on several hosts and the uredinia and telia on a single host. The most extreme type of this class is illustrated by *P. sarcobati* (*P. subnitens*) with over 90 recorded aecial hosts distributed in 22 families with uredinial and telial stage confined to the salt grass, *Distichlis spicata*. In other plurivorous forms, there may be several hosts for both aecial and uredinial or telial stages, or the aecial stage may be confined to one or two hosts while the uredinial and telial stages affect many different hosts which are generally closely related. The development of biological species may be expected in plurivorous forms. The most extreme case of biologic specialization is found in the stem rust of cereals (*P. graminis*) (see discussion of this disease). The existence of numerous biological strains has also been demonstrated in the other cereal rusts: crown rust of oats (*P. coronata*), stripe rust (*P. glumarum*), rye rust (*P. dispersa*), orange leaf rust of wheat (*P. triticina*) and other rusts of less economic importance.

STEM RUST OF GRAIN

Puccinia graminis Pers.

Wheat is affected by three different species of rust: (1) orange leaf rust, *Puccinia triticina* Erik; (2) yellow stripe rust, *P. glumarum* Erik; and (3) stem rust, *P. graminis* Pers. All of these rusts produce a "red rust" stage and a "black rust" stage, and market quotations on wheat in the daily press frequently refer to the severity of "black rust." Rusts are among the most important parasites of our cereals and their distinguishing

characteristics are presented in detail in the author's "Manual of Plant Diseases."

Stem rust was recognized as due to a fungus and first named *Puccinia graminis* by Persoon in 1797. The stage on the barberry was supposed to be an entirely distinct fungus and was named *Aecidium berberidis* Pers., while the uredinial and telial stages were thought to be distinct species, the former being described under the name of *Uredo frumenti* Sowerby. The genetic connection between the uredinial and telial stages was proved by Tulasne (1854), but it remained for the classical researches of De Bary (1864–65) to demonstrate the heteroecism of stem rust and other species. Since these early investigations, the importance of wheat as a world crop has stimulated a steady flow of contributions on various aspects of the disease. Much of the work may be grouped in the following lines: (1) the relation of the barberry to outbreaks of stem rust, with the enactment of laws for the extermination of the barberry (1869 to present); (2) the recognition of specialized strains or races—biological or physiological species (1894 to present); (3) studies on epidemiology in general; and (4) the testing and selection for rust resistance, followed by breeding to obtain resistance.

The stem rust of wheat is present in greater or less amount in practically every country in which wheat is grown, and epiphytotics have been recorded in many countries. The destruction of the barberry has been designed to lessen or prevent these epiphytotics and records show that in many cases they have been prevented or are less frequent. The severe development of the rust is not entirely dependent on the production of the barberry stage, since heavy losses may be produced in certain sections in which the aecial or barberry stage is rare or does not occur, for example, Southern United States, Australia, Central India and South Africa. In the Inland Empire of the Pacific Northwest, the aecial stage is rare and epiphytotics of stem rust have never occurred. The region of greatest severity and economic importance in America is the Great Plains and the Mississippi Valley from Kansas and the Ohio River northward into the Canadian provinces.

Symptoms and Effects on Grains.—The stem rust is characterized by the development of the "red rust" or summer stage on the leaves and other host parts, followed a little later in the summer by the "black rust" or winter stage.

With the onset of the disease, elongated brown or reddish brown granular pustules or *sori* (red rust) burst through the epidermis of any portion of the plant but are especially abundant on the stem and leaf sheath, hence the common name *stem rust*. These pustules may develop without any surrounding chlorotic or dead cells, but in other cases sori or groups of sori are seated in chlorotic areas which soon become dead. The

sori in these hypersensitive areas may be smaller and poorly developed, or in the extreme cases of resistance may show only as minute yellowish or brown "flecks." As the season advances the red rust pustules are

Fig. 112.—Diagrams showing six degrees of rustiness, which may be used in estimating the percentage of rust infection on leaves or stems. (*After U. S. Department of Agriculture.*)

replaced by black pustules or sori (black rust), which occupy the same position or burst through the epidermis at other points. The rust sori may be few in number, or they may be very numerous and may coalesce to form more or less elongated brown or black powdery streaks. Different degrees of severity may be represented by the accompanying diagrams (Fig. 112).

Fig. 113.—Diagrams showing form, size and arrangement of the uredinia of the three rusts of wheat. *A, Puccinia graminis; B, P. glumarum; C, P. triticina.*

The injury from rust may vary from slight to almost a complete crop failure, the damage being caused by: (1) lessened photosynthetic activity and the appropriation of food by the rust pathogen, and (2) the increased water loss due to the rupture of the epidermis by the rust sori resulting in partial sterility, poorly filled heads and shriveled grains. When drought and hot weather coincide with rust attacks, the rusted grain suffers more severely than normal grain or the burning injury is increased. In measurable rust attacks, there is a reduction in both the quantity and the quality of the threshed grain, the reduction in yield lagging somewhat behind the actual percentage of rust. As an example, Marquis in Manitoba in 1937 suffered an 84 per cent reduction in yield, a reduced test weight of 14 pounds and a

lowering of quality from Manitoba No. 1 Hard to Feed Wheat (Greaney, 1941).

If shriveled grain from a badly rusted field is used for seed, it may produce weak plants, a later emergence, a reduced or smaller root system, and plants of lighter weight that show a poor resistance to Helminthosporium root rot (Mead, 1939).

Symptoms and Effects on the Barberry.—The stem rust on the barberry first shows as small, circular, yellowish spots varying from one or two to numerous, upon the upper surface of the leaf. These increase in size to 2 to 5 millimeters or slightly more, become margined with a brighter color or reddish purple and show a central cluster of minute honey-colored pustules on the upper surface (later turning black), from which ooze minute droplets of pycnial nectar. A group of minute cups with ragged or saw-toothed edges, the *aecia* or *cluster cups*, develop on the under hypertrophied surface. Similar hypertrophied cluster-cup lesions may appear on the fruits and fruit pedicels. This stage generally makes its appearance in June or July, the exact time varying in different regions. Injury to the barberry is slight, consequently, the concern which this phase of the disease causes is due to the passage of the rust from the barberry to one of its grass or cereal hosts.

Economic Importance.—Rust takes a regular annual toll in much of the wheat-producing country of the world, but the greatest losses are in years of widespread epiphytotics. The epiphytotic of 1904 was estimated to have caused a reduction in yield of 23 million bushels for Minnesota, North Dakota and South Dakota, some yields dropping to as low as 4 bushels per acre. The reduced yields from stem rust for a ten-year period from 1915 to 1924 inclusive have been estimated to amount to over 379 million bushels for the 13 barberry-eradication states. The year 1916 was an outstanding rust year with an estimated reduced yield of over 180 million bushels. It is claimed that the epiphytotic of 1935 from North Dakota to Texas was the worst since 1916. It was again epiphytotic in the wheat belt in 1937 and 1938 especially in Kansas. Apparently the rust problem has not yet been solved by the eradication of the barberries.

Etiology.—The stem rust of cereals and various wild and cultivated grasses is caused by the heteroecious rust, *Puccinia graminis* Pers., which produces its pycnial and aecial stages on the common barberry, *Berberis vulgaris*, and to a minor extent on some other Berberis species, and its uredinial and telial stages on wheat, oats, rye, barley and about 75 wild and cultivated grasses. Within the morphological species, *P. graminis*, the biological species have been recognized as shown in the table on page 253.

Analysis of a large number of aecial collections from the barberry-eradication area, showed 34.2 per cent *tritici*, 63.7 per cent *secalis* and

2.7 per cent *avenae* and indicate that the barberry is important in perpetuating the physiological forms especially the *secalis* variety. The enormous number of physiological forms has arisen by mutation and probably to a much greater extent by hybridization, as has been demonstrated experimentally.

Variety	Principal hosts	No. of physi-ological forms
1. *P. g. tritici*...........	Wheat, barley and wild grasses	178+
2. *P. g. avenae*...........	Oats and wild grasses	11
3. *P. g. secalis*...........	Rye, barley and wild grasses	14
4. *P. g. phleipratensis*.....	Timothy and weak on oats, rye, barley and some grasses	2
5. *P. g. agrostis*..........	Red top (*Agrostis spp.*)	1
6. *P. g. poae*.............	Blue grass (*Poa spp.*)	1
7. *P. g. airae*............	*Aira spp.* (*Deschampsia spp.*)	1

In the typical life cycle of stem rust the aeciospores from the barberry, if carried by the wind to some susceptible cereal-grass host, germinate and reproduce an infection, *uredinia* soon appearing with the production of numerous *urediniospores*. These spores are scattered by the wind or other agents and spread the trouble on the same host or on other susceptible hosts where new spores are borne, thus being responsible for the extensive spread of the fungus during the growing season. The *telia* produced later in the season form *teliospores*, which remain dormant during the winter and in the spring germinate and produce typical promycelia and sporidia. The sporidia are forcibly abjointed and carried by the wind to the barberry, on which *pycnia* and *aecia* are formed.

The mycelium of the rust fungus develops in the intercellular spaces of the host and obtains its nourishment by rounded or branched *haustoria* which penetrate the cells. This mycelium soon becomes massed beneath the epidermis, organizes a *sorus*, ruptures the epidermis, and the urediniospores are exposed surrounded by the ruptured host cells. Under optimum conditions for growth, uredinia with mature spores may be developed in six to seven days. The mature uredinia are linear, 2 to 10 mm. long, pulverulent, yellowish brown, and develop the ovate, oblong to ellipsoid, echinulate urediniospores, 14 to 22 by 17 to 45 μ, on hyaline stalks. It has been shown that the urediniospores of the different biological forms differ considerably in size and that size is reduced by unfavorable environmental conditions or growth upon a resistant host.

Under favorable conditions of moisture and temperature, urediniospores germinate by sending out two germ tubes from opposite, equatorial

germ pores and, in contact with a susceptible host, form an elongated swollen vesicle, an *appressorium*, over the mouth of a stoma. A fine branch from this vesicle penetrates a stoma, expands into a substomatal vesicle and produces one or more infection hyphae which establish nutri-

Fig. 114.—Spore fruits and spores of *Puccinia graminis*. I, cross section of a barberry leaf showing aecia (*a*), pycnia (*sp*), peridia of the aecia (*p*), upper epidermis (*o*), lower epidermis (*u*), normal thickness of leaf (*x*) and thickness of hypertrophied portion (*y*); *A*, a young aecium; II, section of a telium, showing teliospores (*t*), epidermis of host (*e*) and subepidermal sclerenchyma fibers (*b*); III, portion of a uredinium with urediniospores (*ur*) and one teliospore (*t*). (*A and I after Sachs; II and III after De Bary.*)

tive relations by haustoria, and the mycelium spreads from cell to cell. Large numbers of sori represent numerous infections.

The same mycelium which produced uredinia may give rise to telia later in the season, the teliospores replacing the urediniospores. The teliospores are pedicellate, chestnut brown, oblong-clavate, rounded or attenuate at the free, much thickened end, two-celled, slightly constricted at the cross septum and 35 to 65 by 11 to 22 μ. The teliospores are resting spores designed to carry the fungus over the winter and, under normal field conditions, germinate in the spring but in some tests infection of

barberry has resulted from inoculations with telia from several grass hosts in October and November (Cotter, 1940). The period of dormancy has been shortened by freezing and by alternate wetting and drying to a minimum of 20 days, while 30 to 40 days was fairly common. In experimental tests, teliospores have retained their germinability for four to

Fig. 115.—*Puccinia graminis*. *A*, germinating teliospore (*t*), with promycelium and sporidia (*sp*); *B*, a typical promycelium. (*After Tulasne.*) *C*, a piece of epidermis from the under surface of a barberry leaf with germinating sporidium (*sp*) and infection thread (*i*); *D*, a germinating urediniospore (*u*). (*After De Bary.*)

six years, but did show a weakened virulence to the barberry (Johnson, 1941). In certain regions, for example the southern United States and most areas west of the Rocky Mountains, the teliospores are not viable and thus play no part in the life cycle.

In germinating, each cell of a viable teliospore can give rise to a typical promycelium, which is pushed out from a germ pore located at the tip of the end cell or just below the cross partition of the basal cell. The four sporidia are forcibly abjointed, are blown away by the wind and come to naught unless they fall upon the surface of a barberry leaf.

Temperatures of 12 to 21°C. are most favorable with 26°C. the maximum for the infection of the barberry. On this host the germ tube enters through the epidermis of the upper surface giving rise to pycnia as described under Symptoms, while aecia follow upon the lower surface. It should be noted that the mixing of the pycniospores of opposite sex on the surface of pycnial spots is necessary for the formation of perfect aecia and aeciospores. White pycnia represent a deviation from normal, and white aecia have been produced by mixing the nectar from separate white pycnial spots. The aeciospores, when mature and separated, are nearly globular, 14 to 26 μ in diameter, yellowish orange, with slightly roughened walls, and fall from the cluster cups to be carried away by the wind. They can germinate at once and if they reach a susceptible grass host, the infection hypha can enter the stomata, establish a parasitic mycelium and soon give rise to a crop of urediniospores.

In some heteroecious rusts, the production of the aecial stage is absolutely essential for the perpetuation of the disease, but this does not hold for the stem rust of cereals, although it is believed that the barberry stage is of importance in increasing the severity of infections. In the absence of the barberry stage, there are several possible explanations for the development of the rust on the cereal hosts: (1) from urediniospores overwintered on volunteer cereals, fall seedings or wild grasses; (2) prolonging the period of susceptibility; and (3) increasing the quantity areas; and (4) by persistence on or in the seed.

Although some different findings have been reported, the majority of evidence points to the fact that urediniospores rarely live over in the area north of Kansas and Ohio, but, south of this region, the temperature and rainfall favor successive development of urediniospores. Under these conditions, a supply of spores is produced, and it has been pointed out that they may be carried northward during May and June to the spring-wheat areas within three days' time and be in a viable condition. The length of life of the urediniospores varies with the temperature and the relative humidity of the air, and they are resistant to desiccation and exposure to light. Their resistance shows that they could be carried long distances and still be capable of causing infections, and their dissemination by wind and air has been proved by ground spore traps and by spore traps carried to the upper air by airplanes. From tests carried out, the persistence of mycelium or sori on the seed has not been credited with carrying the stem rust from one wheat crop to the next. There can be no doubt, however, that the movement of spores from the southern localities to the northern areas is a very important means of spreading the rust.

Predisposing Factors.—It is a matter of record that various portions of the world have been visited at various times with epiphytotics of stem

rust of unusual severity; also that rust normally occurs in severe form every year in some sections, while, in others, it is normally present only in traces. These variations in severity must depend largely on climatic factors, with the barberry playing an important role in some regions. In order that an epiphytotic may occur, the following conditions must be fulfilled: (1) a supply of rust spores on the growing grain to give the disease a start; (2) temperature and moisture conditions favorable for germination and infection; and (3) a susceptible condition of the grain at the time spores are being generally disseminated. The first condition may be supplied by aeciospores from barberries, wind-blown urediniospores or overwintering urediniospores; one or several of these sources generally exist. Infection is favored by moderately cool temperatures, abundance of dew, and humid, cloudy or misty days, while sudden showers followed by rapid clearing and evaporation of moisture are unfavorable. If conditions have been favorable for the increase of rust, a great abundance of inoculum may be available by the time the heads have emerged, and the next ten days is a *critical* period, the severity of the rust depending on the conditions which prevail during this period.

In the notable epiphytotic of 1904, the temperatures were subnormal during the *critical period*, and are believed to have favored the development of rust by: (1) increasing the number and rapidity of infections; (2) prolonging the period of susceptibility; and (3) increasing the quantity of inoculum. The importance of temperature and rainfall has been emphasized in the study of the more recent epiphytotics. Studies have shown that when winter wheat headed before the first of June no epiphytotics developed, but, if wheat enters the heading stage later and optimum temperatures prevail with an even distribution of precipitation above normal, an epiphytotic will develop if sufficient primary inoculum is available.

Rust is increased in severity by any practices which produce a heavy stand, or shaded growth such as heavy applications of nitrogenous fertilizers, heavy seeding or an undergrowth of weeds. Potash fertilizers have been shown to increase resistance but sometimes to lower the yield. Spring wheat generally suffers more than winter wheat and late-seeded spring wheat more than early-seeded, primarily because their maturity is delayed until rust has had time to produce a greater abundance of inoculum. This emphasizes the value of fall seeding over spring, or the selection of early maturing varieties rather than late maturing. Early maturity as an escape from rust is well illustrated by Prelude wheat, sixty-day oats, and other quick-maturing cereals.

Varietal Resistance.—It has long been known that varieties of wheat show marked differences in their susceptibility to rust, and it has also been observed that *a variety apparently resistant in one locality may be*

severely rusted in another locality, or that a variety may rust one season but not in another in the same locality. This behavior can now be explained by the existence of biological strains in one locality that are absent from another.

✓In a study in 1922 of 37 biological forms the following host groups were recognized: (1) *immune*—no uredinia, flecks usually present, but sometimes not evident; (2) *very resistant*—uredinia minute and isolated, surrounded by distinct hypersensitive areas; (3) *moderately resistant*—small to medium-sized uredinia, with necrotic circle or halos, or sometimes in sightly chlorotic islands; (4) *moderately susceptible*—uredinia medium, coalescence frequent, no hypersensitive circles, but sometimes chlorotic areas; (5) *very susceptible*—uredinia large, numerous and confluent, entire absence of true hypersensitiveness, but with chlorosis under unfavorable conditions; (6) *heterogeneous*—uredinia very variable, all types and degrees on the same blade. Of the 12 differential hosts used, none were immune to the entire 37 biological forms, but Kanred was immune to 11. Mindum, Arnautka and Speltz Marz were very resistant or moderately resistant to a considerable number of strains, while Vernal Emmer and Khapli Emmer were very resistant to the majority of the biological forms. Later studies have shown marked resistance by two selections of Kanred, Black Persian, Kota and Webster, the latter resistant to more physiologic forms than any other wheat. Marked resistance to stem rust has been shown by several oat varieties including White Tartarian, Raukura Rustproof, Anthony and Minrus.

✓ Many studies have been concerned with attempts to determine the basis of resistance to stem rust and three factors have been suggested: (1) *structural* or anatomical resistance, affected by the size, shape and disposition of collenchyma strands and the compactness and thickness of the cell walls; (2) *functional* resistance, caused by delay in the opening of the stomata and their early closure thus preventing the entrance of infection hyphae; and (3) *protoplasmic* resistance due to physicochemical properties of the living substances. Both structural and functional resistance have been shown to be either of minor importance or not operative, which leaves protoplasmic reaction or properties as the accepted explanation. Various attempts have been made without much success to isolate inhibiting chemical constituents, which points to the conclusion that it is the living protoplasm that offers the resistance.

Prevention or Control.—The prevention or control of stem rust is one of the most difficult problems in wheat production in the areas subject to epiphytotics. The following practices have been emphasized:

1. *Cultural Control.*—Select early maturing varieties which will escape rust; seed early rather than late; use winter wheat where hardy, rather than spring varieties; avoid heavy seeding, poorly drained sites and heavy nitrogenous fertilization.

2. *Dusting with Sulphur.*—Early attempts at control by spraying showed but little benefit, but recent trials with sulphur dusting have afforded protection in experimental trials in Canada and, in extensive operations by the use of airplanes, in California. Sulphur dust has been used at the rate of ℠25 to ℠30 pounds per acre. Yields for oats were increased in Canada from 29.5 bushels to 74.8 bushels per acre in 1930.

3. *Chemical Treatment of the Soil.*—Some promising results have been obtained by chemicals mixed with quartz sand and scattered over the surface of the soil six to seven days after planting but only in experimental tests. It is not likely that this practice will be practical for field culture but may be of value in plot or greenhouse cultures (Hart and Allison, 1939).

4. *Selection and Breeding of Resistant or Immune Strains.*—Some progress has been made but this work is complicated by the large number of biological forms of stem rust and their variable occurrence in different environments. Selection has given some highly resistant strains or varieties (see Varietal Resistance) and breeding has shown that crosses may be obtained that will be resistant to a large number of the biological forms.

5. *Eradication of the Barberry.*—This can be of value only in those areas in which the cluster-cup stage is commonly developed. In some portions of the Old World, the destruction of the barberry has resulted in the elimination of epiphytotics of stem rust. The campaign for barberry eradication in the northern Mississippi area has been in progress since 1918 and millions of bushes have been destroyed. If this eradication of the barberry had not been undertaken, it is certain that this alternate host would have increased in prevalence and thereby would have greatly increased the severity of rust outbreaks. The hope of completely successful control by this method seems doubtful, in the light of the recent (1935) severe development of the disease in the Dakotas, the worst since 1916. Barberries may increase the rust menace by furnishing inoculum to carry the rust back to wheat or other cereal or grass hosts. Physiologic forms have arisen in part by mutation, but probably to a greater extent by hybridization which cannot take place without the barberry stage, and as a breeding ground for originating new physiologic forms.

The common barberry (*Berberis vulgaris* L.) and its varieties and a considerable number of closely related species are susceptible to stem rust, including *B. canadensis* and *B. fendleri*, native species. The former grows wild in the mountains of West Virginia and adjacent states and is of local importance while the latter is of no importance since it grows only in the mountains of southern Colorado. The Oregon grape, *Mahonia aquifolium*, sometimes rusts, consequently, it should not be grown in the barberry-eradication area. The Japanese barberry (*B. thunbergii* DC.) and some of its close relatives are immune to stem rust and may, there-

fore, be grown with perfect safety.   The relation of the barberry to stem rust is presented in detail in U. S. Dept. Agr. Farmers' Bul. 1544, Revised November, 1930.

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APPLE RUST

Gymnosporangium juniperi-virginianae Schw.

This disease has frequently been called the "cedar rust" or the "cedar-rust disease" of apples. The causal fungus spends a part of its life cycle on the cultivated apple or other related hosts and another stage upon cedars (*Juniperus spp.*).

The rust fungus responsible for this disease was first described by Schweinitz in 1822, but it did not assume importance on apples until 1889. In succeeding years it became of more and more importance as an apple parasite, and, since 1905, numerous bulletins and reports have been devoted to various phases of apple rust. The disease is now widely distributed throughout the eastern and central portions of the United States from Maine to Florida and westward to the plains states, but it has attained its greatest severity in the prairie states of the central Mississippi Valley and in the commercial apple districts of Virginia and West Virginia.

Symptoms and Effects on Apple Trees.—The rust affects leaves, fruits, and more rarely the one-year-old twigs. Pale yellow spots first appear on the upper leaf surface, increase in size, and finally become orange-colored with minute black specks (pycnia) occupying the center. In the typical infection, the leaf tissue beneath the spot swells up to form a cushion or blister ¼ to ½ inch in diameter, on which are borne a central group of minute tubular projections enclosing the cluster cups. When mature, the tube splits open, and, when dry, the segments recurve stellately to expose at the bottom a mass of brown or orange powdery spores. With susceptible varieties and a very abundant inoculum, the spots may be so numerous that they remain small, coalesce and cause the leaves to turn yellow and fall before any cluster cups are matured. With a moderate number of infections per leaf, susceptibility may be indicated by the type of lesion: (1) highly resistant show circular, brown spots with pycnia only; (2) slightly susceptible, one to five cluster cups per lesion; (3) moderately susceptible, 6 to 15 per lesion; and very susceptible, 16 or more cups per lesion.

Rust lesions, located at the nodes and involving buds, or even the internodal area, may be formed on the young twigs of susceptible varieties with the development of slightly enlarged cushions covered with the characteristic cluster cups. In susceptible varieties, young fruits may be very generally infected, the lesions being localized or involving the entire surface. Pycnia appear first as in leaf infections, to be followed by the cluster cups on the same lesion but, frequently, surrounding the central group of pycnia. Fruit infections may cause deforming and atrophy or, when mild, only a slight injury.

The injury to the apple from rust may be due to: (1) reduction of functional leaf area and defoliation; (2) reduced set, dwarfing and reduction of quality in fruit from direct infection and from loss of foliage surface; and (3) a reduced vitality which may contribute to winter injury or even carry over into the next season. It has been shown that rusted

Fig. 116.—Mature aecia on the under surface of an apple leaf.

leaves lose control of their transpiration, photosynthesis is materially retarded and respiration is more rapid than in healthy foliage.

Symptoms and Effects on Cedar Trees.—The disease on the cedars causes chocolate-brown, globular, subglobular or reniform corky galls from $\frac{1}{16}$ to 2 inches in diameter. These are first evident in June, but do not reach full size until fall. During the first warm spring rains an elongated, gelatinous orange-colored projection or horn is pushed out, from each circular surface depression. These gelatinous horns dry up and

the old galls are left, finally, as dark-brown, almost black, hard structures which may persist on the tree for some time.

The cedar trees may show only a few galls or they may be so heavily infected that their branches are bent with the weight of the "cedar apples." The injury to the cedar varies with their age and the severity of infection, and in extreme cases may be responsible for their death.

Etiology.—Cedar rust is caused by one of the true rust fungi, *Gymnosporangium juniperi-virginianae* Schw., which completes a part of its

FIG. 117.—A normal fruit and fruits deformed and atrophied by apple rust. (*Drawn from a photograph.*)

life cycle on the apple and a part on cedars (*Juniperus spp.*), the two hosts being required for the perpetuation of the disease.

Three other species of Gymnosporangium may attack the apple: (1) *G. globosum* Farl., the hawthorn rust, also from the red cedar; (2) *G. germinale* (Schw.) Kern, the quince rust, also from the red cedar; and (3) *G. libocedri* (P. Henn.) Kern, from the incense cedar. The first two are found in the eastern United States, the third on the Pacific Coast.

The following is a summary of the life cycle of the cedar-rust pathogen: (1) spore horns, or telial sori are produced from galls in the spring; (2) the teliospores embedded in these germinate during warm spring rains and produce promycelia with secondary spores or sporidia; (3) as the humidity decreases a little (a drop of 10 per cent or more), the sporidia are forcibly abjointed and are blown away by the wind; (4) the sporidia falling on young leaves or fruits of the apple germinate and start infections; (5) after a period of incubation pycnia appear on the upper side of the leaf lesions,

to be followed later by the aecia on the lower surface; (6) the aecia produce the aeciospores, which fall out of the cups and are carried to the cedar, where they germinate and start infections; (7) the host responds by the formation of the characteristic galls which reach maturity in the late fall and are ready to form telial horns in the spring.

The spore horns consist of the elongated gelatinous stalks of the two-celled teliospores, which are pushed out to the surface by the elongation

FIG. 118.—"Cedar apples" or galls (*Gymnosporangium juniperi-virginianae*) on the common cedar. One at the right alive, the other an old gall of the previous season.

of these stalks. The teliospores are oval to acuminate, frequently constricted at the cross septum, 15 to 20 by 46 to 60 μ, and germinate *in situ*, when the sori swell during the warm spring rains. This swelling of spore horns, with germination of the teliospores, may be repeated during periods of rainy weather up to June 1, the sori drying in the interim, so that there may be one to six or more periods of germination before the supply is exhausted. Each cell of the teliospore can grow out into a typically four-septate, hyphalike structure, the *promycelium*, from each cell of which a short projection (sterigma) is formed bearing a secondary spore, or *sporidium*. These sporidia are forcibly abjointed as the telia dry following a rain and are carried away by the wind, when they may fall upon apple foliage, germinate and send an infection thread through the epidermis. After this, the hyphae become intercellular with formation of haustoria.

After a period of incubation, the rust becomes visible on the upper surface, as pale yellow spots, which enlarge, become a darker yellow and show raised specks, the openings of flask-shaped pycnia, in the center.

FIG. 119.—A single large gall with completely expanded gelatinous telia.

These pycnia soon exude a thick orange-colored fluid which contains the *pycniospores.* These function the same as in other heteroecious rusts.

The internal mycelium stimulates the invaded tissue and causes excessive enlargement and multiplication of the spongy parenchyma to form the characteristic cushion or swelling just below the pycnial group. The swellings soon rupture, the aecia protrude and the outer coating or

FIG. 120.—"Cedar apples" of various sizes just beginning to show telia. (*Natural size. Drawn from a photograph.*)

FIG. 121.—Germination of teliospores of *Gymnosporangium juniperi-virginianae*. *a,* typical germination, compact form; *b*, typical germination, elongated type; *c*, promycelia showing deviation from the typical; *d*, teliospore showing direct production of sporidia.

peridium ruptures or splits into segments or rays which during dry weather may be recurved, exposing the aeciospores and giving an open aecium a stellate appearance. The aeciospores are borne in chains within the peridium and are set free as they mature, when they may be carried away by the wind. The mature aeciospores are 21 to 31 by 16 to 24 μ, dark brown, with minutely pitted walls, and, if they reach susceptible portions of cedar trees, they may germinate and produce infections which finally form the typical cedar apples, but these are not evident until the June following the aeciospore dissemination. This means that the pathogen requires approximately twenty-three months from the time of aeciospore dissemination to complete its life cycle.

Conditions Which Influence Infection of Apples.—For apple rust to develop in an orchard, infected cedar trees cannot be far distant, and then the severity of infection will depend on: (1) the location of the trees with relation to the cedars; (2) the weather conditions at the critical periods; and (3) the stage of development of the apple foliage at the time of dissemination of the sporidia. Infec-

Fig. 122.—A group of aecia much enlarged. (*Photo of drawing by Mrs. Venus Pool McKay, Neb. Exp. Sta. Ann. Rept.* **22,** 1909.)

tion has been reported to originate from cedar trees 8 miles distant, but a cedar-free zone of 1 mile around an orchard will afford commercial protection. The use of cedar windbreaks around an orchard produces conditions favorable for severe infections, while elevation or intercepting barriers may modify severity. Spore dispersal when there are many young leaves may be expected to produce maximum infection, in contrast to no infection or only light infection if the leaves are too young or too old. After the period of normal susceptibility, infections may result from wounds made by hail or rainstorms.

Host Relations.—While the telial stage may be produced most frequently on the common red cedar, *Juniperus virginiana*, it has been recorded on 12 varieties of this species and on *J. scopulorum* and *J. horizontalis*. The aecial stage is not confined to the cultivated apple but has been reported on 18 other species of the genus *Malus*, and pycnia have been reported on 12 other species and varieties.

The spread of rust to apple orchards parallels the development of commercial orchards, but it seems probable that the rust first occurred on the wild crab (*Malus coronaria*) in the southern and eastern United States. The final establishment in orchards is believed to be due to the extensive planting of very susceptible varieties, for example, the Wealthy in the plains country of Iowa and Nebraska and the York Imperial in the Virginias.

Many observations have been made on the comparative resistance of apple varieties but reports from different regions have not always been in agreement, possibly owing to physiologic strains of the rust of which four have been recognized in recent studies (McNew, 1938). A recent compilation has recognized the following groups of varieties: (1) very susceptible, 20; (2) susceptible, 25; (3) resistant, 73; (4) very resistant, 18; (5) immune, 2.

Control and Prevention.—The disease may be completely eliminated from an orchard by interrupting the life cycle of the pathogen by the removal of cedar trees. The practices which relate to the cedar are as follows: (1) the destruction of all cedars within a radius of 1 mile around the apple orchard; (2) the avoidance of cedars for windbreaks around orchards or for ornamental plantings within 1 mile of apple orchards; (3) the removal of the cedar apples before sporulation, a procedure practical only when a few small trees are concerned; (4) spraying the cedar has given some protection when the life of the cedar tree was threatened and in recent tests 80 per cent control has been obtained by spraying thoroughly and heavily with Bordeaux 180 in early spring. The production of new galls was prevented, and the development of sporidia by mature galls was inhibited (Marshall, 1941).

In certain states in which cedar rust has been a serious menace to apple production, special laws have been enacted which provide for the destruction of all cedars within the radius of 1 mile around orchards. This practice has been in operation especially in regions like Virginia and West Virginia, where the cedar is native and widely distributed.

The following practices relate to the apple: (1) avoid planting susceptible varieties in the vicinity of established cedars; (2) if, as sometimes happens in the plains country, the cedars are prized more than the apple trees, the latter may be removed; (3) spraying has been reasonably successful in the hands of careful experimenters, but it has proved unreliable as a general farm practice. Fairly good control has been obtained by five to seven applications of commercial lime-sulphur (1–40) and by Linco colloidal sulphur and several different brands of flotation sulphur; but poorer control with Bordeaux and atomic sulphur; and the least effect from dusting. The spraying must begin when the blossom buds are showing color and continue throughout the period of sporidial dissemina-

tion. The applications to be effective must be made previous to the periods of sporidial discharge, since only a few hours are required for infection after the sporidia reach the apple. For this reason the delay of a single day or part of a day in spraying trees may result in little or no protection.

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ASPARAGUS RUST

Puccinia asparagi

This disease of common asparagus attacks all parts of the plant above ground, causing yellowing and blighting of all green parts which develop after cutting has ceased and, thus, much injury in this indirect way to the crown and roots. The common name is appropriate, as severely affected plants are conspicuous from the numerous pustules of the red-rust stage of the pathogen which appear on the stems and cladophylls.

Asparagus rust has been present in European countries since 1805 when it was first recorded. It made its way to America and soon developed with remarkable rapidity as a disastrous disease, following the first report of its occurrence in epidemic form in New Jersey in 1896–1898. By 1900 it had spread until it was common from New England to the Mississippi Valley and from Dakota to Texas. By 1901 to 1903 the disease was epidemic in the important asparagus sections of California. A little later it spread to the Pacific Northwest but never reached as great severity as in the Southern areas. The rust-resistant Washington strains developed by Norton have made possible a profitable growth of the crop. Heavy losses have been suffered more recently in central

Germany (especially Saxony) from 1931 to 1938, where the Washington strains have been very susceptible. In the state of Washington these Washington strains have also shown heavy infections during 1939–1941.

Symptoms and Effects.—Rust is generally first noticed because of the appearance of red spots in the "feathered-out" tops and may appear any time after the tops are well branched. A reddish or rusty powder is readily liberated from these rust spots or sori (red-rust stage). After the first appearance of the rust, if conditions are favorable, there may be a gradual or rapid spread until the tops become generally affected. The rusted tops soon begin to turn to a bright yellow, giving the appearance of premature maturity, the needles fall to the ground and nothing remains but the bare stalks, covered with the rust pustules. By this time many of the pustules have become black in color as a result of the formation of the black-rust stage.

Early in the cutting season stalks that are allowed to grow up will show lighter green, oval patches on the main stem or branches, and sometimes on the needles. These soon develop concentric zones of round pustules which burst through the surface as yellowish cups and scatter an orange-colored dust (aecial or cluster-cup stage). This form may have largely disappeared by the time the red-rust stage appears, but some remnants may have persisted.

The normal handling of an asparagus field with the removal of the marketed crop is a heavy drain upon the crowns and roots, and when cutting stops, the new shoots begin the work of replenishing the depleted food reserve and building for the next season. The development of rust in severe form retards or even checks this photosynthetic activity, and the plants may go into their period of winter rest in a starved condition. As a result, the next season the weakened crowns produce shoots which lack size and vitality, thus affecting both quantity and quality of the product. With each successive attack, the crowns become weaker and either the field becomes unprofitable or the plants may be killed. Weakened crowns may also become invaded by rot-producing organisms which help to complete the work of destruction.

The amount of injury from rust is extremely variable, is influenced by locality, soil and other factors affecting the severity of attack and also depends on the resistance of the variety or strain. Losses as high as 50 per cent have been known to result from a single attack, and losses of 15 to 35 per cent have been common. Conover's Colossal, a very susceptible variety, has been practically exterminated from the Atlantic Coast areas.

Etiology.—Asparagus rust is caused by *Puccinia asparagi* D.C., a true rust fungus which produces all its spore stages on the single host and · in the normal succession: pycnia, aecia, uredinia and telia.

The overwintered, two-celled teliospores which have fallen to the ground germinate in the spring, each cell sending out a promycelium which is typical for the rust fungi, and each gives rise to four secondary spores or *sporidia*. These sporidia are forcibly detached and are carried by the wind or convection currents to the stems of near-by asparagus plants. Infections occur on the stems or branches in the early spring during the cutting season, by the penetration of infection threads through the stomatal openings. The hyphae ramify in the intercellular spaces

FIG. 123.—Portion of a section of a reredinium of *Puccinia asparagi*. (*After Ralph E. Smith, Cal. Bul.* 165.)

FIG. 124.—Portion of a section of a telium of *Puccinia asparagi*. (*After Ralph E. Smith, Cal. Bul.* 165.)

and send haustoria into the cells. Minute yellow *pycnia* appear in groups in the center of the oval, light-green, cushion-like lesions, and the *aecia* soon follow in a surrounding ring or in concentric circles. The aecia are short-cylindric or cup-shaped, with a pale peridium, and are filled with orange-colored aeciospores, 15 to 18 μ in diameter, with hyaline granulose walls. The aeciospores are wind-disseminated and germinate at once and make new infections if moisture is available.

In May or earlier, the uredineal or red-rust stage begins to appear, and from that time on the rust pustules become more and more abundant. The dusty-reddish-brown sori yield enormous numbers of yellowish-brown, globular or oval *urediniospores*, 21 to 24 μ, and provided with four germ pores and yellowish markings on the spore wall. These spores are able to germinate at once, and their widespread dissemination causes a general spread of the rust. They remain viable for a few weeks only, but as they are being constantly formed during the growing season, an abundant supply of viable spores is always prevalent to reinfect any susceptible structures.

Later in the season, or as affected parts become exhausted, the fungus makes provision for its winter rest by the formation of the black-rust, or *telial* stage, which may follow the uredinia on any of the affected parts. By fall the telial pustules are very abundant and prominent. Each telium is a cluster of dark-colored, thick-walled, two-celled spores of the typical Puccinia form, borne singly on long hyaline stalks to which they remain attached. These teliospores vary from ellipsoid to pyriform, are frequently constricted at the cross septum, measure 30 to 60 by 21 to 28 μ, and remain attached to the old host parts, hence are not wind-disseminated. On fallen needles and stems they remain on the ground where they are ready to germinate after their period of winter rest and cause the first spring infections.

It is of interest to note that the rust fungus is parasitized by a number of imperfect fungi, including *Darluca filum* Cast. which produces pycnidia, *Tubercularia persicina* Ditt., which forms sporodochia, and perhaps more rarely by a Cladosporium.

Conditions Favoring Rust.—The severity of rust is modified very much by environmental conditions. (a) In some environments the occurrence of heavy dews is more important than rainfall. Smith (1905) has shown that in the semiarid coast country of California rust increases with the dewfall and that regions which have little or no dew are practically free from rust. This also holds true for the Pacific Northwest. Heavy irrigation which promotes dew formation favors infection, but sufficient must be used to keep the plants in a thrifty condition. (b) Rust is more severe on light, sandy, loam soils, than on heavier soils, especially during dry seasons. It does more damage on peat lands than elsewhere, when in equal abundance. (c) Well-spaced rows (10 feet) running with the wind permit more rapid drying out than rows running across the wind and, consequently, retard the rust, while localities sheltered' by windbreaks or otherwise protected from the sweep of the winds suffer more than those localities subjected to drying winds. (d) Observations on the effect of fertilizers are somewhat conflicting, some reporting but little if any effect, but others claim an increase of rust accompanying potash deficiency (Weise, 1937). The object should be to produce well-nourished, vigorous plants with good recuperative power. (e) Fields abandoned to weeds and suffering for water increase the damage and suffer more than well-cultivated fields supplied with requisite moisture. It is also reported that female plants are especially susceptible to injury.

Host Relations.—The common cultivated asparagus (*Asparagus officinalis* L.) and its varieties are the only crops that suffer from the attacks of this pathogen, although a number of other species may be affected. Early in the history of asparagus rust in this country, the

Palmetto strains or varieties stood out as resistant in comparison with Conover's Colossal, and consequently the former and some related varieties soon predominated in the eastern sections and also in California. The rust problem was not solved because canners were opposed to Palmetto because of its coarseness and color. A possibility was offered of obtaining new and valuable and more resistant varieties by breeding, since preliminary work had shown variations in susceptibility in individuals of a given strain and also revealed the fact that certain European varieties were more resistant than local varieties. As a result of this breeding work by Norton (1913), the industry was saved by the production of the so-called Washington pedigreed strains, Martha Washington and Mary Washington, the former the most highly resistant strain yet found, and the latter sufficiently resistant for profitable culture. Less valuable stock was also distributed under the names: "Washington Stock," "Martha Washington Stock," and "Mary Washington Stock." The rust problem has again come into prominence, since these Washington types do not seem to have maintained their resistance. They have been reported as susceptible in Germany and more recently in Washington, hence there is still work for the breeder.

Control.—Three different measures are available for reducing the losses from asparagus rust:

1. *Sanitary Measures and Cultural Practices.* Abandoned fields and wild plants along roadways and fences should be either dug out or kept cut during the summer. If potash deficiency is found to be promoting the severity of infection, this factor should be corrected (see also (e) under Conditions Favoring Rust).

2. *Spraying.* The first efforts in spraying were not followed by the desired success because the fungicides that were used would not spread and stick to the smooth surfaces of the asparagus stems and also because spraying machinery suited to the crop was not available. The difficulties were somewhat overcome in New York by the use of special spraying machinery and a resin Bordeaux. This method gave only fair protection and was unduly expensive because frequent applications were required. As a result of tests in California, it was shown that sulphur in liquid or dry form gave better results than any other treatment, and preference was given to sulphur dust. The recommendation of Smith (1906) was as follows: (a) an application about three weeks after cutting stops; (b) a second application one month later; (c) another about a month later, using at each time one-half sack of the highest grade *flowers of sulphur* per acre put on in a dusty, smoky cloud, so as completely to cover the tops. More recently spraying with Bordeaux plus an adhesive or with some other copper fungicide has been recommended in Germany (1931–1938) and has been used with varying degrees of success, some workers reporting good control, while others claimed only partial control.

3. Use of the most resistant varieties and breeding for the production of new and resistant strains (see Host Relations).

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BLISTER RUST OF WHITE PINE

Cronartium ribicola Fischer

This destructive disease of white pines is caused by one of the heteroecious rusts which requires the presence of an alternate host (Ribes species) for the completion of its life cycle.

History and Geographic Distribution.—This rust was first reported for North America from Geneva, N. Y., in 1906 on *Ribes*, and in 1909 on the white pine. Infected white pine seedlings from a German nursery had been widely distributed throughout the Northeast, and a year later it was found that infected stock had also been introduced from several French nurseries. This extensive introduction of European-grown stock was due to the removal of the high import tariff and the consequent reduced cost, which stimulated the development of the planting of the white pine. The disease was first found at Vancouver, British Columbia, in 1921, and has spread to the western white pine in British Columbia, Washington, Oregon, and Idaho and to the sugar pine in northern California. In the east the blister rust has spread to the New England states and west to Minnesota and south into Virginia.

Blister rust was first reported on Ribes and white pine by Dietrich in 1856, but was at first thought to represent distinct species. Klebahn

Fig. 125.—Map showing range of five-needled pines and distribution of white-pine blister rust in the United States and Canada. Data on distribution of blister rust in Canada furnished by Dr. A. W. McCallum, Canadian Department of Agriculture. *(From Farmer's Bul. 1885.)*

(1888) and later investigators proved by inoculations that *Peridermium strobi* is the aecial stage of *Cronartium ribicola* on various species of Ribes. By 1900 the disease was widespread over the northern and most of the western portions of Europe which will account for the heavy infection of nursery stock as recorded above. It is the belief that the blister rust was introduced into Europe from Asiatic Russia where it was present on the Swiss stone pine.

Symptoms and Effects.—The first evidence of infection on the pine is the appearance of small golden to reddish-brown spots on the needles, which later become more conspicuous. The infection spreads from the needles to the stem, and the infected bark may assume a yellow to orange color and exhibit a characteristic spindle-shaped swelling of the bark during the second season following infection of the needles, or not until the third season in case of the western white pine. After an interval varying from six to nine months, pycnia appear on the bark "as small, honey-yellow to brownish patches which swell slightly, forming shallow blisters." The bark over these blisters ruptures, and a yellow or honey-colored, sweetish, sticky ooze filled with minute rod-shaped bodies, (*pycniospores*) is extruded. These are washed away by rains or may be eaten by insects, and only pycnial scars are left.

The aecia are formed over the area occupied by the pycnia, making their appearance in the next spring or summer as large whitish or yellowish blisters, rounded to elongate or irregular, $\frac{1}{8}$ to $\frac{1}{2}$ inch in diameter, enclosed by a delicate membrane, the peridium. This soon ruptures and exposes the orange-yellow mass of *aeciospores*, which are disseminated, leaving later only the roughened and fissured bark. The lesion is extended the next year, with the production of more pycnia and aecia, and this sequence is repeated until the stem is completely girdled, the time required depending on the size of the invaded branch. The stem beyond a girdling lesion is killed, and such infections are marked by "flags," or branches showing reddish-brown dead foliage.

The downward advance of an infection makes it possible for it to extend from smaller into larger branches and in many cases finally into the main trunk which may in turn be girdled and ultimately all distal parts will be killed. On eastern white pine no swelling of lesions is visible on stems two or more inches in diameter (sometimes even constrictions), but on western white pine stems up to 5 inches in diameter the lesions may be swollen. A conspicuous flow of resin may be evident from the lesions.

The presence of the pathogen on its alternate hosts, species of currants and gooseberries, is indicated first by the formation of small, pale yellow blisters which finally burst on the undersurface of the leaves and expose the orange-colored uredinial pustules. The tissue of the leaf may be

further invaded and more similar pustules formed, until affected areas are either killed or turn yellow. Later with the maturing of the leaf tissue, the pustules are produced but cause no necrosis and are not visible on the upper surface, while more and more pustules are formed.

FIG. 126.—Ornamental white pines killed by blister rust. (*From Farmer's Bul.* **1885.**)

As the season advances, the orange-colored pustules are no longer produced, but instead small fingerlike or elongated reddish-brown projections or telial columns are formed, singly or in clusters or so abundant as to cover the lesions on the under surface and produce a felted condition that has suggested the name "felt rust" for this stage. In the less susceptible species of cultivated currants and gooseberries, the injury to these hosts is of small importance, but in the very susceptible black currant, the affected leaves may die early in the season, and fall, resulting in much defoliation.

FIG. 127.—Drawing of an infected 12-year-old main stem. *a*, advancing edge of the infection; *b*, the pycnial area; *c*, the aecial area on which the bark is cracked and broken. (*After Colley.*)

Etiology.—The blister rust is produced by *Cronartium ribicola* Fischer, a heteroecious rust, which forms its pycnia (0) and aecial (I) stages on the various species of five-needled pines and the uredinial (II) and telial (III) stages on a large number of species of Ribes generally known as currants and gooseberries. The germination of the individual cells of the telial columns results in the production of the promycelial (IV) stage. The relation of the Ribes and pine stages has repeatedly been demonstrated by inoculations from Ribes to pines and from pines to Ribes (see History and Geographic Distribution) and has been verified by numerous field observations and surveys.

The pycnia or spermogonia appear first on a young lesion and produce large numbers of small, pear-shaped pycniospores which are exuded in honey-colored, sweetish drops on the surface of the bark. These were formerly considered to be degenerate spore forms without definite function but are now believed to have a sexual function. The pycnia are normally developed on the outer portion of an advancing lesion back of the edge of the invasion line and in young infections appear at least one season before the aecia, but after that both may appear simultaneously on adjacent portions of the lesion (see Symptoms).

The whitish or orange-colored masses of aeciospores as noted under the description of symptoms are composed of many individual ellipsoid to ovoid cells, 18 to 20 by 22 to 31 μ, with colorless and coarsely verrucose walls, but with a basal and lateral smooth area. The aeciospores are wind-borne often for great distances and retain their vitality for weeks or even months. On germination, an aeciospore gives rise to an infection hypha and this, if developed on the undersurface of a susceptible Ribes leaf,

may enter through a stoma and establish an infection. After one to three weeks, uredinial pustules may be produced on the under surface, and these on rupture may release the ellipsoid to obovoid, yellowish, urediniospores which measure 14 to 22 by 19 to 35 μ and have a sparsely and sharply echinulate wall. The urediniospores tend to hold together in clumps and consequently are not disseminated far by the wind, but do spread the infection to other leaves or to near-by Ribes species, and the spread may continue, with as many as seven generations of uredinia in a single growing season.

FIG. 128.—Drawing of a median section through a small mature uredinium. *a*, a binucleate basal cell; *b*, a tetranucleate basal cell just before the urediniospore initial is cut off; *c*, a secondary uridiniospore initial which has just been cut off from the basal cell; *d*, the urediniospore initial has divided into a urediniospore and a stalk cell; *e*, a mature urediniospore; *f*, crushed epidermal cells of the host; *g*, the bank of parenchyma-like cells, which encircle the sorus. (*After Colley.*)

In the late summer or early in the fall, on the uredinial lesions or on new ones, telia which become dark brown when mature may develop as slender brown columns, up to 2 millimeters long. Each telial column consists of an aggregation of vertical columns of oblong or cylindric teliospores 8 to 12 by 30 to 60 μ. Mature teliospores may be developed on Ribes from the first of June through the balance of the season and are able to germinate under proper conditions of moisture and temperature. Many of the individual cells of a telial column will give rise to a short four-celled promycelium, each cell of which forms a small, globose basidiospore (sporidium). These sporidia are forcibly detached and are wind-disseminated, and thus some may come to be lodged on the needles of susceptible pines. Since the sporidia are rather delicate, they soon lose their viability when exposed to sunlight or dry air. They may infect

susceptible pines at least 900 feet distant, and certain of the Ribes species may spread the disease to pines to a distance of about a mile.

Hosts.—The blister rust fungus requires hosts belonging to two botanically distinct groups, species of pines and species of currants and gooseberries, for the completion of its life cycle.

The aecial or pine stage of the fungus has been found on the majority of the five-needle or white pines. According to a list compiled by Spaulding (1922) 11 species were known to be attacked in European and Asiatic countries and in America. The eastern white pine (*P. strobus* L.), the white bark pine (*P. albicaulis* Engelm.), the western white pine (*P. monticola* Dougl.) and the sugar pine (*P. lambertiana* Dougl.) have shown their high susceptibility under natural conditions, the last three being even more susceptible than the eastern white pine. It has been pointed out that Himalayan and Balkan pines may be used as replacements for our native white pines because of their resistance, but this does not appear practical because the former is not hardy under severe winter conditions, and the latter has not come up to expectations for hardiness. The basis for hardiness or susceptibility may be either anatomical or physiological. Some of the structural factors increasing susceptibility are the long retention of the needles, large number of stomata on the needles, and thicker bark. Vigorous trees appear more susceptible, or at least suffer more than slow-growing individuals, probably due in part to the production of many more needles, which are the real targets for infection.

Fig. 129.—Drawing of a short mature telial column in which the spores (*a*) have germinated, producing promycelia and sporidia (*b*). (*After Colley.*)

The uredinial and telial stages of the blister rust have been found .to develop on more than 60 species of currants and gooseberries (Ribes) in Europe and America on the basis of inoculation tests. The occurrence

of the Ribes species in a given stand of pines and their degree of infection influences the rapidity of spread and the amount of damage to the pines. The common or European black currant (*Ribes nigrum* L.) grown in many home gardens and the wild black currant (*R. petiolare* Dougl.) of the western white pine region are about equally susceptible with the white-stemmed gooseberry (*R. inermis* Rydb.) only slightly more resistant. The cultivated varieties of red currants are in general resistant and, consequently, cause but little infection of pines. A variety of red currant, the Viking, is very nearly immune, and a strain of the red Dutch currant is reported to be immune in the pine-growing areas of Germany. As far as the native Ribes in North America are concerned, it is concluded from field observations of natural infections and cultural tests that almost any of the native species are sufficiently susceptible to be a menace to susceptible pines.

Economic Importance.—The attacks of blister rust are a distinct menace to the life and productiveness of both the eastern and western white pines and sugar pine. The amount of damage will depend on the number and susceptibility of the Ribes species within or adjacent to stands of susceptible pines, and control of the disease is necessary for perpetuation of commercial stands. The effect on both mature stands and on seedlings and young trees should be emphasized.

Mature stands of pine in which the rust is epidemic develop the disease, but the rate of progress varies and may appear to be slow. Branches may be girdled and killed but when the main trunk is invaded, complete girdling and death of all distal parts is only a question of time. It is reported that western white pines may even be killed by numerous branch infections. A number of recorded cases will serve to illustrate the degree of damage: (1) A stand of eastern white pine of trees seven to ten years old free from rust in 1920 showed 69 per cent killed after ten years; (2) a stand of western white pine, 20 feet in height, showed 90 per cent killed after the rust had developed for 11 years, but were practically all dead after five more years. In older stands under similar environments the mortality will be reduced and the life of the forest prolonged.

In infected stands, seedlings and young trees contract the disease and are killed quickly in contrast to older trees. Year after year the seedlings become infected and succumb, leaving only older trees, and when these mature or are killed, the stand is not renewed. This behavior should emphasize the fact that reforestation of cutover areas becomes difficult, since Ribes species are more abundant and the young pines consequently succumb more rapidly.

Control.—The most important method of blister rust control involves the eradication of Ribes within certain areas in which their presence is a

constant menace and which serve to spread the rust from Ribes species to susceptible pines. In addition to this *local* control, attention should be given to silvicultural practices, treatment of ornamental pines and young, pure-culture plantings, and the possibility of the introduction of exotic resistant species or of the selection of resistant native strains. Biological factors, such as destruction of sporulating cankers by gnawing rodents, or the invasion of blister-rust lesions by other fungi may play a minor part in reducing inoculum, but can hardly be considered of commercial importance. It is worthy of note that blister rust is absent in cities and industrial centers even though both of the hosts are present, probably because of the action of sulphur dioxide or other gases.

1. *Local Control or Eradication of Ribes.*—In the East the removal of Ribes species from white pine stands and from a surrounding zone of at least 900 feet is generally sufficient, except for black currants which should be no nearer than one mile. Hand pulling may be practiced, but after 5 to 7 years the area should be gone over to eliminate any missed plants or new growth, always removing as much as possible of the root system. Complete removal may even require a third inspection.

In the Western blister-rust areas, eradication of Ribes is more difficult because of a larger number of species of diverse habits and many individual plants, the larger size of some species, the rugged topography and the consequent inaccessibility of many of the pine forests. The eradication methods have included a limited amount of hand pulling, but mostly other methods including: (a) grubbing with mattocks and picks for smaller bushes; (b) cutting close to the ground followed by chemical treatment of the stumps with either Diesel oil, ammonium thiocyanate or borax to kill the roots; (c) mass eradication especially for stream-type species by spraying with Atlacide or Atlacide plus a noncombustible filler; (d) spraying with ammonium thiocyanate for certain Ribes species resistant to Atlacide; and (e) the uprooting of dense stands by tractor-power bulldozers, followed by burning of the brush and seeding to grass.

2. *Silvicultural Methods.*—Proper cultural methods will not control blister rust but will aid in reducing the damage, especially in the eastern white pine area. Thinning, pruning and weeding of young stands will improve both quantity and quality of the timber and lessen the cost of control. For new plantings the selected sites should be as free as possible of Ribes species, and plantings should be continuous so that the shade will keep the Ribes from becoming established. Care should be taken to avoid disturbance of the forest duff as this may increase the Ribes by new germinations of buried seeds.

3. *Treatment of Ornamental Pines.*—First thought should be given to the removal of Ribes within the danger zone and to the immediate surgical

treatment of affected trees in home grounds, parks, roadsides and recreational areas. Infected trees can be saved if treated before lesions on the main trunk are too far advanced. Branches seriously infected should be removed and trunk lesions treated by cutting out the diseased bark and a surrounding zone of apparently healthy bark. For several years all the pines should be reinspected for lesions that may have been missed or for others that have become visible.

4. *The Use of Resistant Species.*—Two avenues are open for obtaining resistant species: (1) the introduction of exotic, resistant (five-needle) species to replace native species; and (2) the selection of resistant strains of native five-needle pines. Two resistant exotic species have been considered: (1) the Himalayan, but this is not hardy under severe winter conditions; (2) the Balkan. These species are being tested for silvicultural characteristics, wood properties, and site requirements, but definite recommendations must be deferred until more data are available. The rarity of resistant strains of native species, combined with the difficulty of their propagation and maintenance does not offer much promise that the problem can be solved by selection and breeding.

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IMPORTANT DISEASES DUE TO RUST FUNGI

For key references on these diseases see F. D. Heald. "Manual of Plant Diseases," 2d ed., pp. 811–816.

ENDOPHYLLACEAE

Principal host	Common name of disease	Scientific name of causal organism
Houseleek........................	Rust	*Endophyllum sempervivum* (A. and S.) DeR.
Blackberry.................	Short-cycle rust	*Kunkelia nitens* (Schw.) Arth.

COLEOSPORIACEAE

Goldenrod and pine..	Rust	*Coleosporium solidaginis* (Schw.) Thüm.
Scrub pine........	Leaf rust	*Gallowaya pini* (Gall.) Arth.

CRONARTIACEAE

Five-needle pines................... Ribes spp.	Blister rust	*Cronartium ribicola* F. de Wal.
Pine (27 species)................... Oak (29 species)	Eastern gall rust	*C. cerebrum* (Pk.) H. and I.
Pine (13 species)................... Castilleja, etc.	Western gall rust	*C. harknessii* Mein.
Pine (3 species)................... Castilleja, etc.	Lodgepole blister rust	*C. filamentosum* (Pk.) H. and L.
Pine (*P. ponderosa*)................ Comandra	Pine-Comandra rust	*C. pyriforme* (Pk.) H. and L.
Pine (17 species)................... Myrica and Comptonia	Pine-sweet-gale rust	*C. comptoniae* Arth.
Pine (4 species)................... Ribes spp.	Pinon blister rust	*C. occidentale* H. B. and H.
Spruce........................ Ericaceae	Leaf rusts	*Chrysomyxa spp.*
Spruce........................ Ericaceae	Leaf-blister rusts	*Melampsoropsis spp.*

MELAMPSORACEAE

Grape............................	Rust	*Physopella vitis* (Thüm.) Arth.
Fig.............................	Rust	*P. fici* (Cast.) Arth.
Hemlock.........................	Leaf and cone rust	*Necium farlowii* Arth.
Larch............................ Poplar	Poplar rust	*Melampsora medusae* Thüm.
Hemlock......................... Poplar	Poplar rust	*M. abietis-canadensis* (Farl.) Lud.
Fir............................. Poplar	Poplar rust	*M. albertensis* Arth.
Larch............................ Willow	Willow rust	*M. bigelowii* Thüm.
Flax.............................	Rust	*M. lini* (Pers.) Desmas.
Hemlock......................... Rhododendron	Leaf and cone blister rust	*Pucciniastrum minimum* (Schw.) Arth.
Hemlock......................... Vaccinium	Leaf and cone blister rust	*P. myrtilli* (Schum.) Arth.
Hemlock......................... Hydrangea	Hydrangea rust	*P. hydrangeae* (B. and C.) Arth.
Spruce........................ Rubus spp.	Raspberry rust	*P. americanum* (Farl.) Arth.

Melampsoraceae— *(Continued)*

Principal host	Common name of disease	Scientific name of causal organism
Fir and spruce...................... Caryophyllaceae	Witches'-broom	*Melampsorella elatina* Arth.
Fir............................... Blueberry and huckleberry	Witches'-broom	*Calyptospora columnaris* (A. and S.) Kühn

Pucciniaceae

Bean............................	Bean rust	*Uromyces appendiculatus* (Pers.) Fries
Carnation........................	Carnation rust	*U. caryophyllinus* (Schr.) Wint.
White clover......................	Clover rust	*U. trifolii-repentis* (Cast.) Liro
Red and zig-zag clover..............	Red-clover rust	*U. trifolii* (Hedw.) Lev.
Alsike............................	Alsike rust	*U. hybridum* Davis
Coffee...........................	Leaf disease	*Hemileia vastatrix* B. and Br.
Ranunculaceae..................... Stone fruits	Rust	*Tranzschelia punctata* (Pers.) Arth.
Raspberry and blackberry...........	Orange rust	*Gymnoconia interstitialis* (Schl.) Lag.
Quince, apple, etc................. *Juniperus spp.*	Eastern quince rust	*Gymnosporangium germinale* (Schw.) Kern
Pear, apple, etc................... *Juniperus spp.*	Eastern pear and apple rust	*G. globosum* Farl.
Apple and crabapple................ *Juniperus spp.*	Common apple rust	*G. juniperi-virginianae* Schw.
Apple, pear and quince.............. Incense cedar	Pacific coast rust	*G. libocedri* (P. Henn.) Kern
Snapdragon.......................	Rust	*Puccinia antirrhini* D. and H.
Asparagus........................	Rust	*P. asparagi* DC.
Chrysanthemum...................	Rust	*P. chrysanthemi* Roze
Buckthorn........................ Oats	Crown rust	*P. coronata* Cda.
Cereals and grasses................	Yellow-stripe rust	*P. glumarum* (Schm.) E. and H.
Barberry......................... Cereals and grasses	Stem rust	*P. graminis* Pers.
Sunflower........................	Rust	*P. helianthi* Schw.
Hollyhock........................	Rust	*P. malvacearum* Mont.
Beets, spinach, etc................ Salt grass, etc.	Spinach rust	*P. subnitens* Diet.
Oxalis sp....................... Corn	Rust	*P. sorghi* Schw.
Ranunculaceae.................... Wheat, spelt and grasses	Orange leaf rust	*P. triticina* Erik.
Borage spp...................... Rye	Brown leaf rust	*P. dispersa* Erik.
Star-of-Bethlehem................ Barley	Dwarf leaf rust	*P. simplex* (Koern.) E. and H.
Rubus spp.......................	Blackberry rust	*Kuehneola albida* (Kühn) P. Magn.
Rubus spp.......................	Western or yellow rust	*Phragmidium rubi-idaei* (DC.) Karst.
Rosa spp........................	Rose leaf rusts	*Phragmidium spp.*
Rosa spp........................	Rose cane rust	*Earlea speciosa* (Fries) Arth.

CHAPTER XI

DISEASES DUE TO BASIDIOMYCETES

PALISADE FUNGI

This group includes the most important forms of the true basidium fungi or Basidiomycetes, represented by the familiar toadstools, mushrooms, shelf or bracket fungi, puffballs, earthstars, bird's-nest fungi and

FIG. 130.—Clamp connections of basidiomycetes. *A*, the development of clamp connections. (*After Paravicini.*) *B*, *Stereum purpureum;* *C*, Rhizoctonia; *D*, a whorl of clamp connections in *Coniophora cerebella;* *E*, *Merulius lachrymans* with branch from one clamp connection. (*B–E, adapted from various sources.*)

stinkhorns. The familiar structures which are ordinarily referred to as "the fungus" are, in reality, the fruiting bodies only, while the mycelium or vegetative body of these fungi is hidden within the substratum, and must have made an extensive growth before the fruits or sporophores appear.

The Mycelium.—Distinctive features of the mycelium are: (1) the binucleate condition of the cells of the hyphae; and (2) the presence of characteristic forms of cell unions known as "clamp connections." Uninucleate and multinucleate cells may sometimes be formed.   The cells which give rise to the basidia are always binucleate.   In fully developed clamp connections a slight bulging is noted on one side of the hyphae just back of a cross septum and this appears to overlap the beginning of the other adjacent cell.   A clamp connection is formed by the growth of a short branch just back of a septum, which curves over until its tip comes in contact with the cell on the other side of the septum, when fusion takes

FIG. 131.—Development of the basidia of *Armillaria mucida*.   (*After Kneip.*)

place, bringing the cell contents of the two adjacent cells in communication.   The opening is generally closed later, leaving the characteristic clamp.   The presence of these connections is frequently a convenient means of recognizing a basidiomycetous mycelium.

Spore Types.—The characteristic feature is the production of club-shaped hyphae, the *basidia*, which form the continuous, uninucleate *basidiospores*, on slender terminal outgrowths, the *sterigmata*.   The common or typical number of basidiospores for each basidium is four, but, in some species, the number may be two, six or eight.   The basidia are mostly arranged side by side in a more or less extensive palisade-like layer, the *hymenium*, which rests on a more compact subhymenial layer. Enlarged sterile cells, *cystidia*, may be mingled with the basidia.   Con-

idiospores and chlamydospores are formed in some species but play a minor part in reproduction.

Types of Sporophores.—According to the type of spore fruit, three orders are recognized:

1. Hymenomycetales or Agaricales characterized by variously organized fruits that are open at maturity so as to expose the *hymenium* and permit the forcible separation of the basidiospores. This order furnishes many parasitic or semiparasitic species distributed in the several families and genera as follows:

a. Exobasidiaceae, or the gall-forming palisade fungi in which the hymenium covers the surface of hypertrophied host parts. Includes the single parasitic genus *Exobasidium.*

b. Thelephoraceae, or the smooth shelf fungi, with sporophores varying from a loose mycelial weft to a pellicular, fleshy membranous, tough fleshy or rough leathery structure that is resupinate, rolled, shelving or branched. The following genera furnish some parasitic species: *Septobasidium, Corticium, Coniophora, Stereum* and *Thelephora.*

c. Clavariaceae, or the fairy clubs and coral fungi, with sporophores fleshy or tough, cylindrical or club-shaped and unbranched or branched in coralloid form with cylindrical, flattened or even thalloid branches. The genera furnishing parasites are *Typhula* and *Sparassis.*

d. Hydnaceae or the tooth fungi, with sporophores membranous felt-like, fleshy, corky, leathery or woody and resupinate, shelving or stipitate and with the hymenial surface covering small warts, short spines, flattened or cylindrical teeth or slightly anastomosing toothlike plates. The genera furnishing parasites are *Hydnum, Echinodontium* and *Steccherinum.*

e. Polyporaceae or the pore fungi with sporophores annual or perennial, fleshy, leathery, corky or woody and resupinate, shelving, or centrally or eccentrically stipitate. Hymenium generally in closely packed, cylindrical or angular tubes, but sometimes covering the surface of shallow pits, anastomosing wrinkles or labyrinthiform lamellae. The important genera are *Merulius, Poria, Fomes, Polyporus, Polystictus, Trametes* and *Lenzites.*

f. Agaricaceae or the gill fungi, with sporophores soft fleshy or sometimes waxy or leathery and generally of typical mushroom form, but more rarely sessile or shelving. Hymenium covering the surface of radiating plates, or *gills.* The important genera are: *Marasmius, Lentinus, Schizophyllum, Pholiota, Armillaria and Pleurotus.*

2. Gasteromycetales characterized by closed basidium fruits from which the spores are set free only by the formation of a definite opening, by irregular rupture or by decay. These forms are illustrated by the puffballs and related fungi and are all saprophytic.

3. Phallales characterized by basidial fruits that are at first subterranean, tuberlike structures, which at maturity rupture and allow the

viscid spore mass to be carried rapidly upward by the expansion of an interior mass of elastic tissue. This type is illustrated by the stinkhorn or carrion fungi. A single species, *Ithyphallus impudicus,* is a doubtful parasite on sugar cane.

Palisade Fungi as Agents of Wood Disintegration.—The majority of the palisade fungi of economic importance are wood-destroying organisms, but a few are able to grow on herbaceous substrata. There are three agencies responsible for the destruction of timber while still standing in the forest or after it has been worked into various products. These are fire, insects and wood-destroying fungi, the last working mostly as silent or hidden enemies. The wood-destroying fungi may be purely saprophytic, growing only on dead or structural timber, but, in many cases, these are of as great concern as definite parasites, since they may bring about the disintegration of foundation timbers, posts, poles or any timber which is in contact with moisture. The various wood-destroying fungi which invade living trees are for the most part *wound parasites,* gaining an entrance through pruning wounds, sunscald cankers, winter-injured branches, mechanical injuries from hail, lightning or wind, insect tunnels, basal fire burns or various other injuries which may expose the wood to attack.

Certain species of palisade fungi confine their attacks very largely to the roots or basal part of the trunk and thus bring about their disintegration or cause a *root rot.* Other species make their best development in the heartwood, or in the portion of the tree trunk containing only dead tissue, and are unable to advance into the outer or sapwood. These *heart rots* may so permeate the heartwood as greatly to weaken the mechanical support which it should furnish, and also render the timber of little value for structural purposes. Some species which start in the heartwood may advance into the living sapwood and bark and cause the death or disintegration of the living cells. Other species with more of a parasitic tendency may be able to establish themselves at once in living sapwood or bark, without first growing for a time in dead tissue, thus bringing about disintegration which may be called a *sap rot.* The solution of the different constituents by the invading fungi is brought about by the secreted enzymes or digestive ferments, of which many different kinds have been isolated. Three of the marked changes which result may be noted: (1) the digestion of the lignin, leaving the cellulose; (2) the digestion of the cellulose of the walls, leaving the lignin; or (3) the digestion of the middle lamellae, thus causing a separation of the individual cells. Invaded wood may become discolored, brittle cr soft and punky in accordance with the characters of the changes induced by the invading fungi. Complete disintegration may occur at localized points, leaving pockets which are sometimes filled with white aggregates of mycelium.

THE RHIZOCTONIA DISEASE OF POTATOES

Corticium vagum B. and C.

This widespread disease of the Irish potato has received a great variety of common names such as "black speck," "black scab," "scurf," "russet scab," "Rhizoctonia rot" and "little potatoes" because of the small tubers of the affected plants; "brown stem," "stem rot," "stem

Fig. 132.—Young potato plants showing lesions due to Rhizoctonia.

canker" and "potato-collar fungus" based on the nature of the stem attacks; and "rosette," "leaf roll" and "aerial potato" because of other accompanying symptoms.

Since the first recognition of the disease by Kühn in Europe in 1858, a voluminous literature has accumulated on Rhizoctonia as the cause of disease in the potato and numerous other hosts. It is prevalent in varying degrees of severity in all of the continents and also in Japan, the West Indies and New Zealand.

Symptoms and Effects.—The tubers show very characteristic marks or effects of the disease: (1) superficial black bodies, or *sclerotia*, suggestive of "dirt that will not wash off," but in reality fungous structures varying in size from mere specks to giant forms an inch or more in diameter; (2) roughened or checked areas somewhat resembling common scab and designated as *russet scab;* (3) tuber pits or open cavities formed by the

Fig. 133.—Effects of Rhizoctonia on potato plants. *A*, stems cut off by the fungus; *B*. stem lesion and aerial tubers. (*Photographs by B. F. Dana.*)

corrosion of localized areas of tissue; and (4) a rotting of the tubers, either localized or sometimes spreading to cause a rot resembling late-blight decay or to produce a jelly-end type of decay of elongated stem ends. The occurrence of the sclerotia is very general, while the other effects are found less frequently. Many other tuber abnormalities such as little potatoes, tubers but no tops, some types of aerial tubers, and irregular, branched, deformed, split or cracked tubers formerly attributed to this disease are now known to be caused in large part either by unfavorable environmental factors or by virous diseases.

The symptoms of the disease on the developing sprouts or the aerial shoots vary with the age and the growth conditions and include: (1) elongated, reddish-brown or very dark lesions on the stems below the surface of the ground, very evident in contrast to the whitish or yellowish surfaces of normal stems; (2) young sprouts completely cut back or

FIG. 134.—Corticium stage on stems and leaves of potato.

"burned off" by severe stem lesions, this symptom varying in severity from failure of sprouts to reach the surface to a slow recovery and the final production of nearly normal tops; (3) corrosion and drying or even girdling of stem bases as a result of later infection or slower progress of the invading parasite; (4) superficial, interlacing brown threads, or hyphae of the fungus, covering many portions of roots, stolons or stems, and especially evident on the smooth whitish surfaces; and (5) a white, powdery

crust or coating, faint or very conspicuous, on the green stems just above the ground level and extending up the stem for one to several inches.

FIG. 135.—Potatoes showing numerous sclerotia of average size. (*Photograph by B. F. Dana.*)

FIG. 136.—Potatoes showing giant sclerotia of Rhizoctonia. (*Photograph by B. F. Dana.*)

Other accompanying symptoms of variable occurrence are aerial axillary tubers, and yellowing and rolling of leaves. The "rosette" habit and

"large tops but no tubers" formerly attributed to Rhizoctonia are the effects of virous diseases.

The injury from Rhizoctonia will vary according to the degree to which the various symptoms are in evidence, and according to reports range from almost complete crop failure to no appreciable reduction in yield of table stock. The damage caused by the disease may be due to (1) missing hills and reduced production by plants that do survive; (2) the conspicuous sclerotia, which have a disfiguring effect on table stock and make the tubers less valuable as seed stock; and (3) to the less common effects on the tubers, such as pitting and rotting.

Etiology.—This disease is due to a simple basidiomycetous fungus, *Corticium vagum* B. and C., which in its sterile mycelial stage has been described under the name of *Rhizoctonia solani* Kühn. It has been clearly proved by pure culture inoculations that this parasite is able to form the characteristic stem or stolon lesions and that it can induce a rotting of tubers. At times, however, the mycelium may be present in abundance on the surface of stems, roots and tubers, with little or no evidence of disturbance in the life of the host. It has been shown, however, that Rhizoctonia is not the sole cause of the familiar stem lesions, but that various other soil fungi, acting independently or in conjunction with Rhizoctonia strains, are also causally related. Various effects formerly attributed to Rhizoctonia attacks have been noted in the description of symptoms as caused by some of the virous diseases, and it may be noted further that a killing of the absorbing roots and a blight of the tops at one time credited to Rhizoctonia has also been shown to be caused by a virous disease. The causal relation of Rhizoctonia to tuber pits does not seem to have been definitely proved.

Rhizoctonia may be present on the subterranean portions of the potato as a superficial *mycelium* or in the form of mycelial aggregates, or *sclerotia*, while the sporulating or *basidial stage* develops upon the stem above the soil, on leaf petioles or leaflets close to the soil or sometimes on the surface of the soil. The young vegetative hyphae are colorless, vacuolate, septate at intervals of 100 to 200 μ and invariably show branches that are more or less constricted at their points of origin from the main axis. The hyphae soon become colored and then are evident as a web or weft of yellowish-brown or brown strands, frequently so abundant as to be evident to the naked eye, but sometimes so few as to require microscopic examination for their detection. The most mature hyphae are very dark, rather rigid, the cell walls thicker, the cells of uniform diameter (8 to 12 μ) and the branches arise generally at right angles to the main axes. Some sugar beet strains form more slender hyphae (7.8 to 9 μ). In the organization of sclerotia, denser tufted groups of hyphae are formed with profuse branching, shorter cells of irregular diameter showing lobulated, elbowed or moniliform types. In the case

of sclerotial formation, this type of growth becomes still more compact.

The sclerotia are rounded or irregular, more or less flattened, dark brown or almost black, smooth on the surface and are quite easily detached from the skin. The mycelium may penetrate the tissue below the sclerotia, the invasion being intercellular, and may even develop below the periderm in some cases (Schaal, 1939). The most compact sclerotia

Fig. 137.—*A*, portion of potato skin showing section of a sclerotium; *B*, cells from most compact sclerotial tissue; *C*, cells from loose sclerotial tissue; *D*, distributive hyphae from surface of a tuber; *E*, a basidium and basidiospores of *Corticium vagum*. (*After B. F. Dana.*)

appear in section to be made up of closely grouped cells with few intercellular spaces, a pseudoparenchyma, but less dense sclerotia may show a spongy structure of short irregular "bloated" cells with constrictions at the cross septa.

Under suitable conditions of moisture, the network of brown hyphae which clothes the stem advances upward around its base and forms a felt-like mantle of white fruiting hyphae. Numerous branches produce typical basidia upon the surface of the mantle and a white, powdery appearance is characteristic of the mature sporulating condition. Each basidium produces four sterigmata, each of which produces a hyaline, elliptical or obovate basidiospore, 9 to 15 by 6 to 13 μ.

The basidiospores are forcibly abjointed from the sterigmata and are wind disseminated. They are able to germinate at once and establish

new mycelia either in the soil or in connection with other hosts. The parasite is also spread very generally by the use of potato seed stock that is carrying numerous sclerotia.

Predisposing Factors.—The severity of the Rhizoctonia disease of potatoes is influenced by environmental factors: (1) acid or faintly alkaline soils favor its development; (2) fertilizers like acid phosphate or in some cases sulphur increase the per cent of infected tubers; and (3) temperatures from 15 to 21°C. are especially favorable for attacks, but, above 24°C., the injury is very slight.   For beet strains and stolon strains, affecting beets the optimum temperature is 30°C.   This temperature relation will explain why early planting favors the disease and later planting reduces the injury.

Host Relations.—Many cultivated plants and also common weeds of our fields and gardens are susceptible.   At least four general groups of symptoms may be recognized: (1) the damping-off of seedlings or of cuttings; (2) the stem rots or root rots, or the rotting of root crops; (3) the rotting or blighting of foliage; (4) the rotting of fruits.   *Rhizoctonia solani* ranks with *Pythium debaryanum* as an important agent of damping-off, and is probably more serious in this connection than as the cause of disease in older plants.   The damping-off by Rhizoctonia is not confined to seedbeds but is serious in both garden and field, as may be illustrated by the damping-off of beets and the sore shin of cotton.

Herbaceous stems or roots of various hosts may be affected in much the same way as the potato, while woody stems may sometimes be invaded.   Beans, beets, carrots, carnation, eggplants, peas, radish and sweet potato are some of the more important adult herbaceous hosts. One illustration of foliage invasion may be cited in the case of lettuce, in which the leaves of mature plants were rotted.   Examples of fruits rotted by Rhizoctonia are bean pods, green or ripe tomatoes, eggplants and strawberries.   In most cases the invaded fruits are in contact with the soil and entrance occurs during periods of moist or rainy weather. Most of the hosts are dicots but the disease has been noted on wheat, oats, barley, grasses and onions, among monocots and also on coniferous seedlings.

Biological Strains.—The existence of various physiologic strains of *Rhizoctonia solani* has been recognized, although their separation has not been so clearly defined as in numerous other pathogens.   Six strains were recognized from a study of 15 isolations from potato, bean, lettuce, dahlia, eggplant and Habernaria from California and Missouri, on the basis of cultural and physiological characters and pathogenicity.   Isolations from cotton from Egypt, England, India and United States were all strains of *R. solani*.   Strains from Europe, America and Java showed variation in cultural characters, temperature requirements and parasit-

ism. Low-temperature and high-temperature strains were recognized. In recent tests of 89 isolates from potato from widely separated regions of the United States, none were parasitic on the sugar beet, but some isolates from sugar beet were more virulent on potato than any of the potato strains (Le Clerg, 1941).

Prevention or Control.—The adoption of the following control practices should give the highest percentage of disease-free tubers that can be produced:

1. *Seed Selection.*—The use of sclerotia-free seed lessens the chances of heavy infection and in some cases has given better results than seed disinfection.

2. *Seed Disinfection.*—The common occurrence of seed-borne potato parasites has prompted a general recommendation of seed disinfection (for details, see Potato Scab). The following have been recommended for Rhizoctonia: the standard cold corrosive sublimate, the modified acid-containing mercuric chloride, the hot mercuric chloride, the hot formaldehyde and, more recently, the yellow oxide of mercury. In some regions the use of a superphosphate fertilizer has given an improved control. Too long or too strong steeps may produce severe pitting of tubers, kill eyes or induce delayed germination. Organic mercury dusts and dips have generally been inferior to the inorganic dips. The literature of recent years gives many conflicting reports as to the value of seed treatment for Rhizoctonia, some claiming control and increased yields, while others offer opposing data. From numerous experiments it seems certain that seed disinfection for Rhizoctonia control alone is not sufficiently profitable to justify the expense in many environments.

3. *Cultural Practices.*—Attention should be given to the following: (1) rotation to avoid contaminated soils using a four- to six-year rotation, with alfalfa immediately preceding potatoes; (2) the avoidance of acid fertilizers, and the use of lime or manure unless the prevalence of scab opposes it; (3) late planting to avoid the low temperatures known to favor infection; (4) shallow rather than deep planting; (5) regulation of irrigation, especially avoidance of late heavy applications; and (6) harvesting as soon as mature. A marked reduction of the disease has resulted from the use of a 5–10–5 fertilizer, a rye cover crop or 10 to 12 tons of stable manure per acre.

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THE MUSHROOM ROOT ROT

Armillaria mellea (Vahl.) Sacc.

The mushroom root rot has many other names such as the "shoe-string-fungus rot," "Armillaria root rot," "crown rot," "rhizomorphic root rot," "toadstool disease," and on conifers the name "resin flow" or "resin glut." The "honey agaric," the "oak fungus" or the "shoestring fungus" are names applied to the pathogen.

The mushroom root rot is a disease of considerable importance in European countries, Australia, Japan and the United States. In Europe it has received most attention as a disease of forest trees, while in America it has been more important in fruit crops.

Symptoms and Effects.—The first external symptoms of the disease are the decline in vigor and retarded growth of the plant as a whole or of certain branches. Leaves may remain undersize and scanty, turn yellow and fall prematurely, and branches may die back. In coniferous trees, there may be an abnormal exudation of resin (resinosis) from the base of the tree. This resinosis is sometimes the first external symptom in coniferous trees, but browning and dropping of the needle leaves may be expected to follow. In stone and citrus fruits more or less gummosis may accompany the attack.

Diagnostic indicators are: (1) shiny, dark-brown or black, rootlike or cordlike strands, the *rhizomorphs*, $\frac{1}{25}$ to $\frac{1}{12}$ inch in diameter, which run over the surface of the crown or roots in a branched or anastomosing system, ramify between the bark and wood of old lesions or extend through the soil for some distance from the affected parts; (2) somewhat

fan-shaped, feltlike growths of white mycelium which spread out between the wood and bark or within the bark in decayed parts. While the mycelial character is quite distinctive, the detection of the rhizomorphs is unmistakable evidence of the true nature of the disease (Figs. 138 and 139). The later appearance of the characteristic groups of honey-colored toadstools around the crown of the affected plants will give final proof of the presence of mushroom root rot. The fruiting bodies do not always appear and are generally not in evidence until the host is dead or in the

Fig. 138.—Rhizomorphs and young sporophores of *Armillaria mellea*. (*Photograph by H. H. Whetzel.*)

last stages of decline, hence main reliance in diagnosing the disease can be placed on the presence or absence of rhizomorphs.

A susceptible host once infected is doomed unless the parasite can be removed or checked in its advance. On some hosts the progress of the disease is very rapid, and fatal results follow the same season or the season following the initial infection, while in other cases (older forest trees) the tree may make a hopeless struggle through a period of years in a crippled condition. The disease may cause injury by: (1) the localized attacks on roots, followed by their death; (2) the partial or complete girdling at the crown; and (3) the reduced surface extent of the foliage, stunted and poorly matured fruits, and very scanty growth during the progress of the disease.

In orchards or in pure stands of timber, the affected trees generally appear in spots or groups, but where the disease is of long standing and many foci exist, the affected areas may merge more or less, thus obscuring the points of origin. It has been claimed that more trees die in Europe from the attacks of Armillaria than from any other parasite.

In one area 80 to 90 per cent of the Scotch pine were killed in 15 years. In almond and citrus orchards in California, it is noted that the centers of infection frequently coincide with places where oak trees formerly stood, the fungus spreading to the fruit trees from the remains of the old oak stumps or roots. Prune orchards in western Washington, planted on cleared land on which the native growth had been affected by the root rot, suffered heavy losses as illustrated by the death of 583 trees in one orchard of 1053 trees during a period of seven years (1895 to 1901).

Etiology.—The mushroom root rot is due to *Armillaria mellea* (Vahl.) Sacc., a common and widely distributed gill fungus, which produces

Fig. 139.—Rhizomorphs of *Armillaria mellea* between the bark and wood of larch stem. (*After Hiley.*)

sporophores of the Agaricus or toadstool type. This fungus is able to lead a saprophytic existence on stumps and roots of dead trees, but, under favorable conditions, it may become a serious wound parasite. In the case of potatoes, roots of citrus, and various other hosts, the rhizomorphs may penetrate directly into healthy tissue. Trees suffering from unfavorable environmental factors which lead to localized root killing show an increased severity of the disease. The disease is favored by light acid soils but causes less injury in heavy alkaline soils.

This fungus has no conidial stage and relies entirely on the *rhizomorphs* and the *basidiospores*, produced in enormous numbers by the sporophores, for its dissemination. Two types of rhizomorphs are formed, *subcortical* and *free* or superficial. The former replaces the felted mycelium between the bark and wood, when the tree is dead and the bark becomes loosened, and consists of flattened whitish stands which become colored when the separation of the bark exposes them to the air. They branch more profusely than the free rhizomorphs and some branches

enter the host tissue, especially through the medullary rays. The free or subterranean strands or rhizomorphs have an apical growing region and are cylindrical instead of flat and consist of a brown cortex of closely compacted fungous tissue enclosing a central medulla of hyaline hyphae arranged in longitudinal rows.

The sporophores are produced in groups or clusters (rarely single) growing either from rhizomorphs or from cortical mycelial sheets, and

Fig. 140.—Group of nearly mature sporophores of *Armillaria mellea*. (*After Neger.*)

generally appear around the trunk of the host close to the ground level, but in some cases may arise 6 feet or more up a trunk as in the Scotch or Austrian pines. These sporophores are found mainly during the few months of the year preceding the frosts of winter (September, October, November), but occasionally at other times (October to February in California); and are ephemeral structures lasting only until the spores have been disseminated. Each sporophore consists of a *stipe*, 3 to 10 inches long, honey-yellow or brown, bearing the expanded pileus which is honey-colored and sprinkled with dark-brown scales. The lower surface is occupied by the whitish gills (flesh-colored or dingy with age) which are somewhat decurrent on the stipe, which may be with or without an *annulus*.

The pileus is at first distinctly convex, especially in the center, but with age becomes flat or even concave with an upturned margin and

is exceedingly variable in diameter (2 inches to 1 foot). The hymenium consists of paraphyses and an abundance of *basidia*, each generally bearing four sterigmata (rarely two or three) with hyaline, elliptical or slightly reniform basidiospores, 6 by 9 μ. These spores are forcibly detached and fall from between the gills, where they may accumulate as a white powder

Fig. 141.—Small portion of basidial surface of *Armillaria mellea*. (*After Hartig.*)

or be borne away by air currents, the period of spore fall probably lasting several days.

Sporophores may be produced very abundantly from old decaying roots and stumps and from living hosts in the last stages of decline, but, in some cases, only mycelium and rhizomorphs are formed without any fructifications. It is said that sporophores are rare in dense, dark, coniferous woods.

The spores are believed to be responsible for mycelium which develops on dead but uninfected stumps, from which rhizomorphs may spread to adjacent living hosts. It has been pointed out that wood-boring insects are important agents in the spread of the disease and in the establishment of infections. In California the pathogen may be spread by flood waters bearing rhizomorphs, fragments of dead wood or even sawed lumber and boxes (Hewitt, 1936).

Pathological Anatomy.—The mycelial fans or felts may be developed in the cambium, also in the outer phloem or in the inner cortex, and, in such cases, several layers of the mycelium may be found, one inside another. The fungus does not advance far above the crown of the tree, the height varying with the host and the supply of moisture, from 2 to 5 feet or even more. From the cambial felts, the hyphae grow outward into the phloem and inward into the wood—by following the medullary rays and spreading from these into the adjacent tracheides or vessels— and produce a white rot or disintegration of the wood.

Early in the wood invasion black lines or layers appear. Various explanations have been offered by different workers as to their origin: (1) produced at the point of contact of two different invading fungi; (2) bladderlike cells of the parasite, tinted black, and closely crowding the cavities of the tracheides, and also causing some host-cell discoloration; (3) represent the periphery of pseudosclerotia which penetrate to the bark and give rise to the characteristic flattened mycelial strands. Wound gum is formed on the borders of lesions of some hosts, especially walnut and cherry plum; also gum pockets in Prunus species. Seven different enzymes have been obtained from the rhizomorphs and these together with mechanical action bring about host penetration.

Host Relations.—Mushroom root rot is of importance as a disease of forest or shade trees, attacking both coniferous and broad-leaved species. It has been noted especially on cedars, firs, hemlocks, larch, pine and redwood and some other evergreens and on alder, beech, birch, walnuts, tea, avocado, almond, apricot, chestnut, locust, maple, mulberry, oak, sycamore and poplar and other broad-leaved trees, also on azaleas, rhododendrons, hops and dogwoods. The wild hazel has been reported as a favorite host. Two different strains have been recognized: one on conifers, especially Douglas fir, which is not parasitic on apple trees; and the other originating from the native oak, *Quercus garyana*, which causes virulent infections on orchard trees but is not parasitic on conifers.

In America it is of most importance as a parasite of tree fruits ; apples, apricots, peaches, pears, plums, prunes and cherries all suffer. Almonds, citrus fruits, especially oranges, olives, grape, bush fruits and English walnut are seriously affected especially in certain localities. The pear is claimed to be the most resistant of the tree fruits, the French pear being practically immune. Two other deciduous trees are reported as immune, the northern California black walnut (*Juglans californica hindsii*) and the fig (*Ficus carica*).

The potato, carrot, parsnip, rhubarb, dahlias, cannas and strawberries may be mentioned as herbaceous hosts. Rhizomorphs may penetrate potato tubers and develop white, convoluted mycelial plates within the interior. One occurrence of this trouble on potatoes in Washington was in the first crop on new land that had just been cleared of hazel brush.

Control.—In Europe the recommendation is made that infested wood lots be cleared and devoted to the growth of some farm crop for a time and that coniferous plantations should·not follow immediately on land from which broad-leaved trees have been cleared. In America the disease in fruit trees and other cultivated hosts seems to be due largely to planting in cutover land in which the fungus was already established on the native trees or shrubs. In selecting sites for orchards, newly cleared

lands may be viewed with suspicion and planted to a nonsusceptible crop if Armillaria is known to be present.

When the disease appears in established fruit or ornamental plantings, the following practices should be given consideration: (1) The removal and destruction of diseased trees or plants, including as much of the root system as possible as soon as the disease is discovered. This practice should be followed at least when the host is seen to be very seriously affected. (2) The removal and destruction of sporophores when in the button stage, so as to prevent the maturing and dissemination of basidiospores. (3) The construction of barriers to confine the rhizomorphs and keep them from reaching other tree roots. The common barrier suggested is a trench 1 foot wide and 2 feet deep, with the dirt thrown toward the center, surrounding a single tree or groups of diseased trees, and so located as to be beyond the spread of the roots. In citrus orchards in California it is recommended that the affected trees within the trench-enclosed areas should be killed by carbon disulphide poured into spaced holes, 8 to 14 inches deep, spaced 2 feet apart (Hewitt, 1936). (4) The treatment of diseased trees, when the disease is discovered in its earlier stages, with the idea of either saving the tree or prolonging its life. This method consists of removal of the soil from around the crown and the cutting out of diseased bark or roots, either with or without the use of disinfectants and waterproofing, to be followed by a prolonged aeration of the exposed crown. This has given some success with apples in Oregon and with citrus trees in California. (5) Delay of three years in resetting of root-rot spots, or places from which individual diseased trees have been removed, or replanting with a highly resistant variety.

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IMPORTANT DISEASES DUE TO PALISADE FUNGI

For key references to these diseases see F. D. Heald, "Manual of Plant Diseases," 2d ed., pp. 852–857.

1. EXOBASIDIACEAE

Principal host	Common name of disease	Scientific name of causal organism
Cranberry.	Rose bloom or Massachusetts false blossom	*Exobasidium oxycocci* Rost.
Cranberry.	Red leaf	*E. vaccinii* (Fcl.) Wor.
Azalea.....	Galls or hypertrophy	*E. azaleae* March
Vaccinium.	Galls	*E. parvifolii* Hot.

2. THELEPHORACEAE

Numerous.....................	Rhizoctonia disease	*Corticium vagum* B. and C.
Fruit trees....................	Leaf blight	*Corticium stevensii* Burt
Fruit trees and other trees.......	Silver leaf	*Stereum purpureum* Pers.
Apple, pear and some forest trees.	Canker	*Septobasidium pedicellatum* (Schw.) Pat.
Coniferous seedlings...........	Smothering disease	*Thelephora laciniata* Fries
Structural timber..............	House fungus	*Coniophora cerebella* A. and Sch.

3. CLAVARIACEAE

Wheat, barley, rye, beets, cabbage, etc.	Seedling blight	*Typhula* spp.
Fir, spruce, pine and larch............	Yellow root	*Sparassis radicata* Weir

4. HYDNACEAE

Maple and beech..........	Uniform, white sapwood rot	*Hydnum septentrionale* Fries
Oak....................	Wet heartwood rot	*H. erinaceus* Fries
Fir....................	Fir Hydnum	*H. abietis?*
Cotton and many others...	Texas root rot	*H. omnivorum* Shear
Fir, spruce and hemlock....	Stringy, red-brown heartwood rot	*Echinodontium tinctorium* E. and E.
Swamp cedar.............	Top rot	*Steccherinum ballouii* Bank.

5. POLYPORACEAE

Foundation timber, etc..........	House fungus	*Merulius lachrymans* Schum.
Coniferous timber and lumber....	Timber rot	*Poria incrassata* (B. and C.) Burt
Various deciduous trees..........	Common white wood rot	*Fomes igniarius* Fries
Ash...........................	White heartwood rot	*F. fraxinophilus* Peck
Various deciduous trees..........	White butt rot	*F. applanatus* Fries
Conifers.....................	Red heart rot	*F. laricis* (Jacq.) Murr.
Catalpa, etc...................	Yellowish wood rot	*Polystictus versicolor* Fries
Deciduous trees................	Sap rot	*P. pergamenus* Fries
Mountain ash..................	White rot	*P. hirsutus* Fries
Various fruit, nut and shade trees.	White rot	*Polyporus squamosus* (Huds.) Fries
Deciduous trees................	Brown checked wood rot	*P. sulphureus* Fries
Pine and other conifers...........	Red-brown root and butt rot	*P. schweinitzii* Fries
Conifers..........	Brown rot	*Trametes pini* (Brot.) Fr.
Coniferous timber..	Dry rot	*Lenzites sepiaria* (Wulf.) Fr.
Date palm.........	Decline	*Omphalia pigmentata* Bliss *O. tralucida* Bliss

6. AGARICACEAE

Sugar cane.......................	Root disease	*Marasmius plicatus* Walker
Cacao...........................	Witches'-broom	*M. perniciosus* Stahel
Various trees.....................	Scaly cap rot	*Lentinus lepideus* Fries
Shade, nut and fruit trees; also sugar cane.	Schizophyllum rot	*Schizophyllum alneum* (L.) Schr.
Maple and other deciduous trees.	White-streaked sapwood rot	*Pleurotus ostreatus* Jacqu.
Numerous.....................	Mushroom root rot	*Armillaria mellea* (Vahl.) Sacc.
Deciduous trees and conifers.....	Brown-mottled rot	*Pholiota adiposa* Fries

Fig. 142.—Cross section of the trunk of a living silver maple rotted by *Fomes igniarius*. (*After von Schrenk and Spaulding, B. P. I. Bul.* **149.**)

FIG. 143.--Portion of trunk of mountain ash with sporophores of *Polystictus hirsutus*.

FIG. 144.—Portion of trunk of a young cherry tree invaded by *Schizophyllum alneum.* The fungus entered through the long stubs left in pruning.

CHAPTER XII

DISEASES DUE TO IMPERFECT FUNGI

FUNGI IMPERFECTI

In the great groups of fungi many species produce at least two types of spores or spore fruits in the course of their life cycle, as follows:

Fungi	I	II
Black molds and allies (Zygomycetes).......	Zygospores	Sporangiospores or conidiospores
Downy mildews and allies (Oomycetes)......	Oöspores	Conidia or swarm spores
Powdery mildews and allies (Perisporiales)..	Perithecia	Conidia of the Oidium type
Sphaeriales and allies (Pyrenomycetes)......	Perithecia	Conidia of various types
Cup fungi and allies (Discomycetes)........	Apothecia	Conidia of various types
Smut fungi (Ustilaginales).................	Chlamydospores	Conidia rarely
Rust fungi (Uredinales)....................	Telia	Uredinia and aecia
Palisade fungi and allies (Hymenomycetes)..	Basidium fruits	Conidia of various types

Perfect and Imperfect Stages.—The spores or spore fruits indicated in the first column are considered the *perfect stages* by which it is possible to assign a fungus to a definite family or order in the great groups of fungi. The spore forms or spore fruits indicated in the second column represent the *imperfect stages*. These are sufficiently characteristic in some groups to show positively the perfect stage with which they are connected, but, in many forms, the imperfect stages alone offer no certain clue to the perfect stages to which they are related. In such cases the uncertain forms are kept together for convenience as the *Fungi Imperfecti*, a group which may be considered only as a temporary resting place for forms the affinities of which are not known or cannot be safely predicted. It is probable that certain fungi have lost their power to produce the perfect stage, while, in a great number of cases, a perfect form exists but the relationships have not been discovered.

A few illustrations may be cited of conidial or imperfect stages of well-known pathogens for which the ascigerous stages have been discovered in comparatively recent times:

Host	Common name of disease	Imperfect stage	Ascus stage
Stone fruits	Brown rot	*Monilia spp.*	*Monilinia spp.*
Pear and quince	Leaf blight	*Entomosporium maculatum*	*Fabraea maculata*
Apple	Black-spot canker	*Gloeosporium malicorticis*	*Neofabraea malicorticis*
Cherry	Leaf spot	*Cylindrosporium spp.*	*Coccomyces hiemalis*
Plum	Leaf spot	*Cylindrosporium spp.*	*C. prunophorae*
Rubus spp.	Anthracnose	*Gloeosporium venetum*	*Plectodiscella veneta*
Rose	Black spot	*Actinonema rosae*	*Diplocarpon rosae*
Cereals	Scab	*Fusarium spp.*	*Gibberella saubinetii*
Cereals	Snow mold	*Fusarium nivale*	*Calonectria graminicola*
Apple	Black rot	*Sphaeropsis malorum*	*Physalospora malorum*
Apple	Bitter rot	*Gloeosporium fructigenum*	*Glomerella cingulata*
Cotton	Anthracnose	*Colletotrichum gossypii*	*G. gossypii*
Grape	Black rot	*Phyllosticta labruscae*	*Guignardia bidwellii*
Pear	Leaf spot	*Septoria piricola*	*Mycosphaerella sentina*
Iris	Leaf spot	*Heterosporium gracile*	*Didymellina macrospora*
Apple	Scab	*Fusicladium dendriticum*	*Venturia inaequalis*
Barley	Stripe	*Helminthosporium gramineum*	*Pleospora gramineum*

Classification.—Four large subdivisions of the imperfect fungi are recognized as follows:

1. Hyphomycetales.—On the basis of conidia and conidial fruits, four groups with the rank of families are recognized:

a. Moniliaceae.—Conidia from an undifferentiated mycelium, or on specialized conidiophores, single, fascicled or grouped into extensive layers. Mycelium and conidia generally clear and hyaline.

b. Dematiaceae.—Same as Moniliaceae, except hyphae and conidia typically dark.

c. Stilbaceae.—Spore fruit typically a *coremium* (see Fig. 24 *I*). Of minor importance as plant pathogens.

d. Tuberculariaceae.—Spore fruit typically a *sporodochium* (see Fig. 24, *S*, *T*, and *U*). In families *c* and *d* some species may produce fruits characteristic of the Moniliaceae or Dematiaceae when grown on certain substrata. Fusarium is the most important genus.

2. Melanconiales.—Spore fruit typically an *acervulus*. On certain media the Hyphomycetous type may be assumed (see Fig. 24, *O* to *R*).

3. Sphaeropsidales.—Spore fruit a *pycnidium*. This order includes four families but the majority of the plant pathogens are in the *Sphaerioidaceae* (see Fig. 24, *T* to *N*). Gloeodes, Entomosporium, Dothichiza, and Sporonema are some of the more important genera from the other families.

4. Mycelia-sterilia.—Forms for which no spore stage is known. Rhizoctonia, Sclerotium and Ozonium are illustrations of such generic names.

CORKY SCAB OR ACTINOMYCOSIS OF THE POTATO

Actinomyces scabies (Thax.) Güssow and other species

Various other common names have been applied to this tuber disease, such as "common scab," "potato scab," "brown scab," "Oöspora scab," "American scab" and "deep scab." This disease is not to be confused with several other potato troubles to which the name "scab" has been applied, for example, powdery scab (*Spongospora subterranea*), black scab or Rhizoctonia scab (*Corticium vagum*) and silver scurf (*Spondylocladium atrovirens*) sometimes called "silver scab."

The scab organisms are believed to be native to the soil and appear to exist in nearly all soils, especially in those which are well cultivated and rich in humus. The parasitic nature of the disease was suggested as early as 1842, but it was not until 1890 that the causal organism was isolated and described by Thaxter. Scab continues to be a favorite subject for study as evidenced by the publication of over 60 articles since 1935.

Symptoms and Effects.—The scab lesions on the tubers may show as slightly raised or bulging, roughened, corky areas, or these may be nearly on a level with the remainder of the potato skin, or a corky depression may occur. The lesions may be small and few in number or numerous and larger and produce a general infection with little or no normal surface remaining. Some observations and experiments have shown a tendency for the scab lesions to be segregated on the "stem-end" portion of the tuber, except that this condition is the least evident when potatoes are subjected to the temperatures at or near the optimum for scab development. Based on symptoms and causal organism, as many as six different types of true scab lesions have been recognized, the three most common being designated common, deep and russet scab. The normal symptoms may be modified by the work of some of the other tuber diseases (black scurf or Rhizoctonia, powdery scab, skin spot, silver scurf or wart) or by the work of flea beetles, wireworms, white grubs, and mites or millipedes, which frequently extend and deepen the lesions.

The effects of scab are: (1) *The lowering of quality.* This results from (a) the disfiguring effect and the losses when prepared for cooking; (b) an objectionable earthy odor or taste; (c) increased shrinkage in storage; and (d) increased liability to storage decays. (2) *Reduction in yield.* A reduction equal to one-fifth to one-sixth of a normal crop has been recorded from the use of untreated, heavily scabbed seed.

Etiology.—Corky scab of potatoes is caused by various species of Actinomyces, probably the most common and important species being *Actinomyces scabies* (Thax.) Güssow. This organism, first isolated and

Fig. 145.—Corky scab of potato. (*After Lutman and Cunningham, Vt. Bul.* **184.**)

proved to be pathogenic by Thaxter, was named *Oöspora scabies*, and later transferred to Actinomyces. Other species of Actinomyces are able to cause scab: 7 described by Wollenweber (1920); 11 by Millard and Burr (1926); and 21 strains by Wingerberg (1933). The number of distinct strains or species is uncertain, but the numerous pathogenic forms offer one explanation for the various types of scab lesions. According to some studies the type of lesion is not constant for a given strain, but may vary with the age of tubers, variety and place of origin (Noll, 1939). It is generally agreed, however, that *A. scabies* is the most common form.

The scab pathogens are still considered by bacteriologists as filamentous bacteria belonging to the order Actinomycetales, but botanists are now in general agreement that they represent simple forms of imperfect fungi (Hyphomycetes). The hyphae are very slender, 0.5 to 1 μ in diameter, branched, wavy or curved, irregularly segmented and form either aerial or immersed hyphae, or simple short conidiophores, which produce chains of globose or ovoid, nonseptate, hyaline conidia.

The scab lesions originate by the penetration of the pathogen through young lenticels, with stimulation of the meristem and the infection and collapse of deeper layers of cells until the invaded area is deepened and broadened and finally is cut off by a cork barrier.　In recent studies it is claimed that the pathogen may also enter through stomata and in small tubers at points where two cells join.　The cells are split apart by the digestion of the middle lamellae by the action of the parasite which may penetrate the entire tuber (Lutman, 1941).　The author's illustrations of

FIG. 146.—*A*, drawings of *Actinomyces scabies* showing branching and irregular segmentation; *B*, groups of conidia; *C*, section of an old scab.　(*After Lutman and Cunningham, Vt. Bul.* **184.**)

this behavior are not entirely convincing.　In some forms of scab only one cork barrier is formed, but, in others, the organism may break through the first cork barrier and stimulate the formation of other layers of protective cork.

The scab organisms grow and multiply rapidly in soil rich in humus or containing manure, or in the compost heap, but they are of rather universal occurrence in soil, even in raw desert land of the western United States.　Since they survive passage through the digestive tract of animals and grow well in compost, the organisms may be spread by dumping scabby stock on the manure heap or by feeding scabby tubers to farm animals.

Predisposing Factors.—The amount of scab which develops in any planting, assuming that the organisms are either present in the soil or

introduced with the seed, will depend upon: (1) *Physical properties of the soil.* Scab is more prevalent on light sandy or other well-aerated soils, than on firm compact soils. (2) *The soil reaction.* In general, scab is absent or light in decidedly acid soils and is most severe in a slightly acid or an alkaline habitat, although the hydrogen-ion exponent is not an absolute criterion. The behavior will vary with the soil type and the strain or species of scab organisms involved. Some cases of a scab-free crop are reported at a pH of 5.2, while others record some scab even at 4.0 to 4.3 and maximum infection at a pH of 6.0 to 6.6 with less at greater alkalinity. (3) *Chemical composition.* Scab is favored by the addition of barnyard manure or wood ashes or decreased by the addition of potash salts such as Kainit. The increase of scab caused by the addition of lime is due entirely to the increased alkalinity. The effect of manure may be due to the introduction of an increased number of organisms or to supplying organic matter. (4) *The soil moisture.* Both low and medium soil moisture favor scab, the maximum infection occurring at about 40 per cent, while higher moisture may yield a clean crop. The moisture factor is most effective in producing scab while the tubers are still young (about 2 centimeters in diameter). (5) *The soil temperature.* This factor may affect the number of tubers infected, the number of lesions per tuber and the size of the lesions. The temperature range of 17 to 21°C. is optimum for number of lesions and above 20°C. for size of lesions and about 23°C. for the highest per cent of scabby tubers, while the range of development has been given as 11 to 30.5°C. (6) *The soil microflora.* The competition with other soil organisms will reduce the amount of inoculum and thus reduce the amount and severity of the infections.

Host Relations.—While corky scab is primarily a potato disease, it is known to affect beets, carrots, turnips and radishes. On these hosts, the scab lesions frequently form one or more girdling zones, suggesting the name "girdle scab."

Resistance of potato varieties to scab has been attributed to (1) the thickness of the skin; (2) close-textured and partly buried lenticels; (3) suberized lenticels; and (4) to inherent physiological factors; the last is considered of most importance by some investigators. Resistant varieties have been reported by both American and European studies. One general statement is that russet types show greatest resistance, the semirussets medium resistance, while the white and thin-skinned varieties are the most susceptible, with a range from 98 per cent of the tubers scab-free to none scab-free.

Prevention or Control.—The following practices have been shown to be of value: ·

1. The use of clean or scab-free seed, preferably certified stock.

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PEACH SCAB

Cladosporium carpophilum Thüm.

The disease of the peach which affects fruits and to a less extent leaves and twigs has been called peach scab, also freckles and black spot. These common names have been employed because of the effects on the fruit, which are most evident and destructive, while the leaf and twig lesions are of minor economic importance.

Since the first report of the disease by Von Thümen in Austria in 1877, scab has been reported from most of the states from New Hampshire to Florida and west to Texas, Oklahoma, Kansas and Nebraska, and less

frequently from Idaho, Washington and California. It has not been prevalent in the important central peach-growing districts of Washington. The disease is known to occur in various foreign countries including Europe, South Africa, Australia and South America although it is of much less importance than in the United States. The peach scab fungus was collected by Earle in Illinois on fruits in 1881 and on leaves in 1887, by Langlois in Mississippi on fruits in 1886, and even earlier by Galloway. The first report of peach scab as a disease in America was by Arthur (1889), who recorded its presence in Indiana. Later the disease was studied by Taft (1894) in Michigan, by Sturgis (1897) and Clinton (1904) in Connecticut, by Selby in Ohio (1898 and 1904) and by Scott (1907, 1908, 1909) and associates (1910, 1911), the later publications giving especial emphasis to control by spraying. The most complete account of the disease was published by Keitt in 1917, but most of the more recent studies have dealt primarily with control and with host specialization (Bensaude and Keitt, 1928).

Symptoms and Effects.—Peach scab is most conspicuous on the fruits causing superficial spots or lesions, but less evident on the twigs and leaves.

Fruit Lesions.—The fully developed fruit lesions are evident as superficial, circular, olivaceous to black areas, 2 to 5 millimeters in diameter, which may be few in number or so numerous as to coalesce and sometimes cover one-half or more of the fruit surface. Young infections are evident as imperfectly defined, greenish to olivaceous spots up to ½ millimeter in diameter, but these gradually enlarge to form the mature lesions as already described. The infections are generally most abundant adjacent to the peduncle and on the upper portion of the wettable surface, while the protected surfaces may remain free from infection. The fungus is superficial, or invades only a few layers of superficial cells which are cut off from the deeper normal cells by a layer of cork cells. The presence of the fungus results in inequalities of growth, especially in early infections, between normal and diseased areas, and growth stresses may cause small cracks extending into the flesh, or in heavy infections the cracks may be large and even extend to the pit. Heavily scabbed fruits do not ripen uniformly, frequently have a poor flavor and if lesions are adjacent to or involve the peduncle may drop prematurely.

Twig Lesions.—These lesions by the end of the growing season appear on the green or year-old twigs as "smooth, irregularly oval, light-brown to dark-brown areas of normal elevation, with slightly raised purplish to dark-brown borders, which usually vary from ½ to 1 millimeter in width and shade off peripherally into the color of the surrounding normal surface." (Keitt.) These lesions are elongated parallel to the axis of the twigs and may vary in size from 3 to 5 by 5 to 8 millimeters. The twig

lesions may enlarge slightly during the second season, but by the third summer they are less evident and gradually fade out. The lesions when numerous may become confluent and form extended affected areas one or several centimeters in length. By the end of the first summer conidiophores and conidia are produced sparsely, but during the next season they may form conspicuous olivaceous tufts.

Fig. 147.—Elberta peaches badly affected by scab. All show cracking and the upper right fruit an infection by brown rot. (*After Keitt.*)

Leaf Lesions.—The spots on the leaves appear on the under surface as angular to irregularly circular areas, mostly 1 to 2 millimeters in diameter, varying from light brown to purplish. Along the midrib and petiole, the lesions may be longer and narrow and ½ to 1 millimeter wide by 3 to 8 long. The sporulation, similar in character to that on the twigs, is confined to the lower surface of the lesions, but older lesions cause pale yellow or purplish areas on opposite portions of the upper surface.

In considering the effect of scab, main attention should be focused on the injuries to the fruit which are responsible for a lowered market

value. Scabbed fruit is lowered in grade (1) by the surface spots or freckles which render it less attractive; (2) by unevenness of ripening and by lack of uniformity in size and shape; (3) by the cracking of the skin and splitting of the flesh; (4) by impaired flavor in heavy infections; and (5) by the increased injury from bacterial and fungal invasions, especially brown rot. Before adequate control measures were practiced, many localities reported losses ranging from 60 to 25 per cent, and as late as 1911 (Scott and Quaintance) it was estimated that the disease was

FIG. 148.—Section of the skin of a peach fruit made through a scab spot, showing the groups of dark-colored fungus cells from which the conidiophores arise, and also the long, spike like hairs which cover the surface of the peach. (*After Owens*.)

responsible for a loss of 10 per cent of the total value of the crop in the eastern United States.

Etiology.—This disease of peaches is caused by *Cladosporium carpophilum* Thüm., one of the imperfect fungi.

The young hyphae are hyaline, branched and septate and develop beneath the cuticle of the affected organs, but soon become dark or olivaceous. Transverse and longitudinal divisions occur with the result that pseudoparenchymatous masses of fungous cells are developed beneath the cuticle of fruits and stems and with rupture of the cuticle become exposed to the surface. When grown on a suitable culture medium similar stromatoid masses of hyphae may be formed as well as moniliform mycelial threads. *Conidiophores* of varying height and one to several septate but more or less flexuous grow up from the surface

lesions and produce conidia acrogenously. As conidia are formed the conidiophore elongates, and the points of attachment of the conidia are marked by wartlike processes or by bends.ᷣ On different organs, and under varying conditions, the conidiophores may vary in diameter from 4.2 to 5.5 μ and from 30 to 35 μ in length to 90 to 100 μ, especially on the fruit lesions, and may be formed singly or in tufts and in this case may be short and thick. The conidia range in size from 4.2 to 5.2 μ in diameter and from 14.4 to 17.2 μ in length and are produced singly or in short chains (rarely branched). They are mostly ellipsoidal, continuous and pale brown, but some are one-septate and slightly constricted in the middle.

The pathogenicity of the peach scab fungus has been demonstrated by successful inoculations on fruits, twigs and leaves, infection resulting regardless of the origin of the spores from any particular organ, that is, it was transmissible by twig or leaf strains to fruits, and by fruit strains to twigs or leaves. The period of incubation on the fruit has been shown to vary from 42 to 77 days, but sometimes infections are evident a little earlier. Infections on leaves and twigs require a shorter incubation period or 25 to 42 days, and secondary infections on these organs may develop even late in the summer. Conidia may be formed on the affected organs as soon as lesions are evident and may continue to be formed as long as favorable conditions prevail.

According to tests with fresh or young conidia in prune decoction, germination ranges from 60 to 99 per cent, but it has been shown that viability decreases rapidly with age, especially under unfavorable conditions. This behavior is of minor significance, since fresh crops of conidia are formed as soon as conditions become favorable for infection.

Dissemination of the conidia involves their detachment from the conidiophores and their transport from point of origin. The conidia in a dry or humid atmosphere are not detached from the conidiophores, but are readily separated from the conidiophores by contact with liquid moisture. On drying they are not borne away by air currents, which indicates that meteoric water is the main agent of transport. Water that drips or spatters from sporulating lesions on affected organs will carry a load of spores. A slight amount of spore dissemination may result from the detachment of spores by rubbing together of parts by the wind and by abrasion from wind-borne sand particles. If insects or birds play any part, it is of very minor consequence.

Varietal Susceptibility and Hosts.—The different workers have reported marked differences in varietal susceptibility. Some of these differences appear to be due to early maturity before the disease has had time to gain headway, rather than to real resistance. Late maturing varieties are in general the most severely infected, since opportunities

for infection are increased, unless adverse weather conditions prevail. Estimates have been made of the comparative degrees of injury based on the decreased market value of the unsprayed crop. The percentage of loss in bad seasons is considered approximately double that of average seasons. On the basis of bad seasons, the following groupings may be made:

Resistant (up to 30 per cent loss): Early Crawford, Hale Early, Hiley, St. Johns.

Moderately susceptible (30 to 60 per cent loss): Alexander, Belle, Carman, Champion, Edgemont, Late Crawford, Stevens, Stump and Waddell.

Very susceptible (above 60 per cent loss): Bilyeu, Chairs, Elberta, Heath, Mountain Rose, Oldmixon, Reeves, Rivers, Salaway, Smock and Tennessee.

The same fungus or a very similar form attacks the apricot, nectarine, plum and cherry in addition to the peach. According to Bensaude and Keitt (1928) the Cladosporiums of stone fruits fall into two groups: (1) those from peach (*A. persica*) and plum (*P. americana*) referred to *Cladosporium carpophilum* Thüm.; and (2) those from cherry (*P. cerasus*) referred to *Cladosporium cerasi* (Rbh.) Sacc. These two forms differed in the thermal range for germination of conidia and for vegetative development, in the form of the germ tubes and of young thalli developed in cultures and in the size of the conidia.

Control.—Three different practices have been considered for the control of scab: (1) the use of the more resistant varieties, (2) orchard sanitation, and (3) spraying. The first two measures are of minor importance, although they should not be overlooked entirely. It is now generally conceded that the pruning out of diseased twigs is impractical as a means of reducing inoculum but that some value may result from pruning in such a way as to facilitate the free access of air and sunlight and thus contribute to the effectiveness of spraying.

In most regions brown rot must also be controlled and in some curculio also. When scab alone is to be controlled, the time of sprayings are (1) about one month after the petals fall, (2) three weeks later, and (3) about one month after the second application. The second and third applications are needed for the midseason and late varieties, the first for the early varieties.

The early studies showed that both standard Bordeaux mixture and lime-sulphur were effective in controlling scab, but they were objectionable under most conditions because of spray injury. The following sulphur fungicides have been used: (1) self-boiled lime-sulphur, 8-8-50; (2) dry-mix lime-sulphur consisting of sulphur 100 pounds, hydrated lime 50 pounds and 6¼ pounds dry calcium caseinate, using 32 pounds for

200 gallons of spray; (3) a finely divided wettable sulphur, either home-made or commercial. The homemade requires 5 to 6 pounds of 300–325 mesh sulphur, 4 to 5 pounds of hydrated lime plus the wetting agent to 100 gallons. According to Hurt (1937) the following wetting agents may be used: fish-oil soap, glutrin, molasses or goulac, at the rate of 1 pint per 100 gallons, except the latter at the rate of 1 pound per 100 gallons. Some of the effective commercial wettable sulphurs are Kolofog, Flotation, Mike, Micronized, Sulcoloid and Crown. (See also Brown Rot.) Dry-mix lime-sulphur has largely replaced self-boiled lime-sulphur because of its ease of preparation and its adaptability for storage.

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✓ EARLY BLIGHT OF POTATO

Alternaria solani (E. and M.) Jones and Grout

This disease has generally been designated as "early blight" to dis-tinguish it from the late blight or Irish blight (*Phytophthora infestans*), but it is known also as "leaf spot" and "Alternaria blight."

The disease was first reported of economic importance in the United States in 1891 and has since been recorded from Mexico to Canada in North America, from all the other continents and from Java, Bermuda, Australia and New Zealand. It probably occurs nearly everywhere that the potato is an important crop.

Symptoms and Effects.—The disease is characterized by the appear-ance of dark-brown or almost black, more or less circular dead areas upon the leaflets, which usually show a concentric series of rings or ridges giving the lesions a "target-board" effect. Under certain conditions, the spots may remain small and more or less angular, but without the target-board effect, being limited by some of the smaller veins. The spots may be few in number and small or they may be numerous and involve a large part of the leaf area, owing to the coalescing of adjacent spots. Affected leaves may show ragged margins or irregular breaks, or, some-times, perforations due to the breaking away of the dead tissue and some-times more or less curling or rolling. In severe cases the final result

may·be a complete blighting of the affected leaves, frequently preceded by the yellowing of the tissue between lesions. The stalks of seriously affected plants may turn yellow and dry up, and brown lesions may also appear on these. In severe cases in late stages of the disease, most of the lower leaves may be blighted leaving only a few green-spotted leaves at the tips of the shoots.

Fig. 149.—*A*, potato leaflet showing leaf spots due to *Alternaria solani; B*, leaf spots due to *Cercospora concors.*

In an epiphytotic of the disease in Bermuda in 1923, the onset was sudden, its progress rapid and the devastation extreme, while the leaf lesions were large and the stem lesions large and water-soaked.

The leaf phase of the disease should not be confused with other leaf troubles of the potato such as Phytophthora blight, Cercospora leaf spot, tipburn and hopper burn, arsenical poisoning, sunscald or necroses of leaf tissue accompanying virous diseases.

The earlier studies recognized only the leaf phase of the disease, but in more recent times (since 1925), a rot of tubers has been shown to result from the inroads of the same organism. Entrance may occur through wounds, but sound tubers may develop the trouble in storage. Tubers from affected fields may even show no evidence of the disease when harvested but later develop severe storage rot. During the last few years the tuber-rot phase of the disease has been reported from

European countries with losses to the extent of 25 per cent of the crop in Holland.

The leaf phase of the disease alone may cause heavy losses. In some areas or seasons, it has been reported to cause more injury than late blight. The injury from epiphytotics is sometimes very great, but the yearly toll is considerable with only mild attacks. With severe foliage injury, the tubers may remain small, immature, soft-skinned, deficient in starch and will not hold up well in storage. Various estimates of losses in the northern Mississippi Valley have ranged from 10 to 25 per cent.

Etiology.—Early blight is caused by *Alternaria solani* (E. and M.) Jones and Grout, one of the imperfect fungi. The organism was first described as a *Macrosporium* but was assigned to Alternaria when it was discovered that spores were sometimes formed in chains, especially when grown in cultures.

The fungus produces a light-brown or olivaceous septate mycelium ramifying in the intercellular spaces and also penetrating cells within the diseased tissues. After the invaded tissue has been killed, brown, septate, erect or ascending conidiophores, 50 to 90 by 8 to 9 μ, emerge from the stomata or push out to the surface (either upper or lower) between the dead cells. Each conidiophore gives rise to a single spore, this arising as a bud or outgrowth from the terminal cell. The spores or conidia are 120 to 296 by 12 to 20 μ, 5 to 10 cross septate, but with longitudinal septa few or lacking, and are "obclavate, brown, terminating in a very long hyaline septate beak (frequently branched) equaling fully one-half the length of the spore." If the spores are washed off from a spot a new crop will be produced under favorable conditions, as many as three or four successive crops being recorded. They are able to germinate at once under favorable conditions of moisture and temperature, with 5 to 10 germ tubes if the temperature is rather low, or the number may be reduced to two or three. Infection may take place through stomata or directly through the epidermal walls or insect injuries may serve as avenues of entrance.

No perfect or ascigerous stage has ever been found, either on over-wintered leaves or in cultures. The source of the inoculum for the first infections in an early crop is supposed to be either the overwintered spores or new spores developed from mycelium that has persisted in the soil or in the plant remains from a former crop. Infected seed tubers also should not be overlooked as a source of inoculum. That the conidia and mycelium may suffice to carry the fungus over the winter is evidenced by the fact that dried diseased leaves have yielded the pathogen in cultures after 12 and 18 months, and that conidia showed a 10 per cent germination after 17 months at room temperature. Early infections

may produce spores to be carried to later maturing plants, wind and insects being the principal agents of dissemination. Under favorable conditions, the period of incubation is relatively short, incipient spots showing 48 to 72 hours after inoculation while spore production occurs

Fig. 150.—Diagrammatic representation of an early-blight spot showing effect on tissues and the production of spores. (*After Rands, Wis. Agr. Exp. Sta. Res. Bul.* **42**.)

within three or four days. The fungus shows a marked tendency to saltate, and physiological strains have been recognized varying in cultural characters and in pathogenicity.

The foliage appears to be most susceptible after the plants have passed their period of greatest vigor and are being weakened by external conditions or by drains of tuber formation. The severity is influenced

by rain, temperature and soil relations, but optimum conditions include relatively high temperatures alternating with moist periods in combination with retarded or reduced vigor of the host. The optimum temperature for spore production in vitro is 26.1°C. with minimum of 1.5° and maximum of 34.5°.

Host Relations.—Tomatoes and eggplants may be infected by the early blight the same as the potato. Not only the foliage but also the fruits of the tomato may be attacked, especially in the southern states and when shipped to northern markets. It has been reported to cause losses of 50 per cent in Louisiana. The disease has been recorded on various species of the nightshade family besides potato, tomato and eggplant, including the common black nightshade, the garden wonderberry, white henbane, black henbane and the apple of Peru.

Variations in the susceptibility of potato varieties have been noted, but most of those showing resistance have been those of poor commercial

Fig. 151.—Germination of spores of *Alternaria solani.* (*After Rands, Wis. Agr. Exp. Sta. Res. Bul.* **42.**)

qualities. The McCormick variety was proved to be the most resistant of 15 varieties with which it was compared, but it is of poor quality so is of main value as a promising parent for the breeding of new resistant varieties. In Great Britain the Golden Wonder is reported as apparently immune.

Control.—As the basis for a rational control, it should be noted: (1) that the pathogen lives over winter in the debris from preceding crops; (2) that the disease is primarily a foliage trouble, resulting from purely localized infections which originate from wind- or insect-borne spores; (3) that tuber infections originate from spores produced on diseased tops. Crop rotation is of importance, and, in case of continuous cropping, all dead vines should be raked together and burned immediately after harvest, but these practices must be regarded only as aids to the control by spraying.

It has repeatedly been demonstrated that both early and late blight can be controlled by spraying with Bordeaux (see Late Blight). In Rhodesia, where early blight is the important disease, increases in yield of 16 to 57 per cent have been reported from the use of Bordeaux, and in Georgia, where the late blight is also important, increased yields have resulted from the use of Bordeaux. Calcium arsenate 1 pound to 50 gallons, with a caseinate spreader, 30 to 40 days after planting has given

good results in Mysore. It should be recognized that there are many localities in which early blight is not sufficiently frequent or serious to justify the expense of spraying.

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CERCOSPORA LEAF SPOT OF BEET

Cercospora beticola Sacc.

This disease is present in most of the regions where beets are grown and has attracted major attention on garden beets and sugar beets, but attacks also mangel-wurzel, Swiss chard and some other hosts. Under artificial conditions in greenhouse cultures the disease has been transmitted to 30 or more species in 12 different families and has been recorded under field conditions from at least six different species. The disease was first recorded by Kühn as early as 1858 and is now known to occur in most of the regions where beets are grown. It was studied in the United States by Pammel in Iowa in 1891, by Halsted in New Jersey in 1895 and by Duggar in New York in 1899. More detailed studies were made later by Pool and McKay (1913, 1916), and the disease came into greater prominence again from 1928 up to the present, especially in European countries.

Symptoms and Effects.—The disease is first in evidence as small brown, nearly circular spots on the leaf blades and on the leaf stalks. As these become older, they show a more distinct outline, and the margins are very commonly tinged with a reddish brown or purple coloration. As the lesions become older, ⅛ inch or more in diameter, the centers assume an ashy gray color, and dark conidial tufts may be visible. The lesions may be few in number or so numerous (several hundred) as to coalesce and form larger dead areas, and in the most severe cases the leaf may be killed. Lesions may also be formed on the stems of the inflorescence, on pedicels of the flowers and on the seed balls. The first infections are on the basal leaves, and as these shrivel and fall to the ground, the next older leaves are invaded and in turn suffer the same fate. The killing of successive crops of leaves causes an elongation of the crown as new leaves are formed, and this more slender neck, bearing the scars of the fallen leaves, has suggested the name "pineapple disease."

The injury from leaf spot is fourfold: (1) The tonnage is reduced. Especially in early attacks, the loss of much of the chlorophyll-bearing

leaf tissue may retard the development of the roots and thus reduce the yield. In later attacks the loss in tonnage may be only slight. (2) The sugar content of the roots may be reduced. Early attacks may be out-

FIG. 152.—Sugar-beet leaf showing typical leaf spot. (*After Wenzel.*)

grown and cause little reduction in sugar content, but late attacks which continue the injury until late in the growing season or until maturity may cause a marked reduction in sugar content. (3) An increased tare

at time of harvest. Since the beet-sugar companies will not use the elongated crowns these must be cut away and are a loss as far as tonnage is concerned. (4) A reduced feeding value of the crowns and leaves. The injury to the crown and foliage may decrease both the quantity and quality of the product which is a valuable feed, especially for dairy cows. It is estimated that in many cases 50 per cent of the feeding value has been lost.

Etiology.—The common leaf spot of beet is caused by the imperfect fungus, *Cercospora beticola* Sacc., for which no ascus stage is known.

Fig. 153.—Conidiophores and conidia of *Cercospora beticola.* (*After Duggar.*)

The internal, intercellar septate mycelium produces substomatal aggregates of cells from which the nonseptate slightly flexuous conidiophores emerge in groups upon the upper surface. The conidiophores give rise to elongated obclavate to needle-shaped, many-septate, hyaline conidia, 3.5 to 4.5 μ in diameter, but ranging in length from 20 to 360 μ. The optimum temperature for spore formation is about 29°C., the minimum 15°C. and the maximum 37°C. For the growth of the fungus the cardinal temperatures are minimum, 5°C.; optimum, 29 to 30°C.; and maximum, 40°C. The length of the conidiophores and the spores has been shown to vary in different collections and from different regions but appears to be governed largely by the temperatures and the humidity which prevailed, high humidity and favorable temperatures for growth yielding the maximum sizes. As the conidiophores grow in length and new conidia are formed and shed, noticeable geniculations are left, consequently the conidiophores are not straight but slightly flexuous.

The conidia when detached from the conidiophores may be spread by wind, splashing rain, insects, and man or animals in cultural operations. They can germinate at once with the production of one to two germ tubes from one or more cells and produce new infections. The conidia have been reported to retain their vitality for five or more months under field conditions and thus are responsible for new infections on the crop of the next season. The conidia require 99 per cent relative moisture for best germination. The germ tubes or hyphae enter the stomata of the upper leaf surface by hydrotropic stimulus according to Schmidt, (1928), but purely by accident according to Vestal (1933). Since

infection is always through stomata, it is most common on mature leaves and can occur only when the stomata are open. The influence of moisture, light and temperature on the reaction of the guard cells determines in part at least the number of infections.

It has generally been stated that only the older leaves of growing plants were infected, but it has been shown by artificial cultures that seedling infection is more easily produced than infection of mature plants (Vestal, 1933).* Inoculations by dusting with conidia from pure cultures or from powdered, infected leaves were effective under greenhouse conditions in producing lesions on hypocotyl and seed leaves, in some cases sufficiently severe to cause damping-off. It seems probable that similar infections may occur on seedlings in the field.

Early attempts to grow the leaf-spot fungus in cultures were successful but without sporulation. Later studies have been successful in inducing sporulation, a number of media giving an abundance of spores, but beet-leaf agar and potato-dextrose agar have been very satisfactory and have yielded conidia within 36 to 72 hours of growth. Different isolations on a given medium have shown cultural variation but no definite physiological races have been recognized. The use of sporulating cultures for inoculations has made possible rather extensive tests which have shown that the fungus under greenhouse conditions can infect numerous weeds that grow in the sugar-beet areas, and a smaller number under field conditions. On some of the weed hosts the fungus continued to grow saprophytically on the old dead and completely necrotic leaves, and it has been shown that spores from such sources are able to initiate typical infections on the beet.

Control.—The leaf spot cannot be eliminated by any preventive practices, but it can be reduced by attention to the following: (1) *Cultural practices.* Since the fungus lives over on the infected remains of the host left in the field, these should either be removed or buried by deep fall plowing. It is also important to practice crop rotation, as it is claimed that an interval of two years with a nonsusceptible crop will reduce the inoculum to a negligible amount. Attention should also be given to the elimination of susceptible weeds. Favorable conditions for sporulation and infection can be reduced by wide spacing, for example, 20-inch check rows, thus allowing a free circulation of the air and a consequent reduction in humidity. (2) *The use of clean or disinfected seed.* If the seed is infected, it should be treated with one of the mercury dusts or with sulphuric acid (Stolze, 1931). (3) *Spraying or dusting.* It has been demonstrated that leaf spot can be almost perfectly controlled and yields increased by spraying, but the cost of spraying has been shown in many cases to exceed the value of the protection gained. In general, dusting has not been as uniformly effective as spraying. The

fungicides used have been Bordeaux (4–4–50) and copper sulphate-lime dust (20–80) with three to five applications, with best control with the maximum number. (4) *Use of resistant varieties.* Some progress has been made by the recent release of the variety U.S. 200 × 215 which is reported to be highly resistant.

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BEAN ANTHRACNOSE

Colletotrichum lindemuthianum (Sacc. and Magn.) Bri. and Cav.

This disease, known most frequently as "bean anthracnose," is also called bean "spot disease," "speck," "pod canker," "pod spot" and "rust," the latter name being frequently used by gardeners, although the name should only properly be applied to troubles caused by true rust fungi.

The disease was first discovered in Germany in 1875, although it occurred previous to that time in various parts of Europe. Since that time, it has become world-wide in its distribution, having been reported in either moderate or severe form. In the United States, it has reached its greatest severity in the states east of the Rocky Mountains but is either rare or entirely absent in the Pacific Coast states.

Symptoms.—The anthracnose affects seed, seedlings, leaves and other vegetative parts, but it makes its typical and characteristic development on the pods.

FIG. 154.—Anthracnose on pods of beans. Pod at the left completely covered with anthracnose lesions.

The disease appears on the *seed* as yellowish, brownish or blackish spots, varying in size from mere specks to lesions involving half of the surface, frequently quite evident on the white-skinned varieties but generally very obscure on the dark-skinned seed. The lesions fre-

quèntly extend through the seed coat and involve the cotyledons and, on germination, enlarge, become depressed and develop sticky spore masses.

Lesions may occur on all aerial vegetative parts including stems, leaf blades and petioles, flower pedicels, bracts of the inflorescence and sepals. On young seedlings the spots are generally below or at the point of attachment of the cotyledons, but on older plants the black or dark brown lesions are more scattered over the different parts of the stems. In the extreme cases, the dark stem lesions may coalesce to form large lesions 3 or 4 inches in length and, in very young plants, may be so serious as to have a damping-off effect. In leaf infections the lesions appear mostly on the veins and petioles, sometimes affecting the latter so seriously that they cannot support the leaf blades. Leaf tissue adjacent to the infected veins may wither and turn brown.

The first evidence of the disease on the pods is the appearance of minute brown or rust-colored spots which rapidly enlarge, from mere specks to spots 1 centimeter or more in diameter, and become sunken at the center and darker in color, while a rusty-brown, hazel-colored or even reddish border persists around the outside. Spots may be few or so numerous as to coalesce and form irregular or extended lesions. During moist weather, pink, sticky spore masses may accumulate on the sunken, ulcerlike spots. Some lesions may be confined to the wall of the pod, while others penetrate to the seed and are responsible for the symptoms noted for mature seeds.

Effects and Economic Importance.—Bean anthracnose causes losses due to (1) poor stands from low viability of infected seed or from death of affected seedlings; (2) reduced yields; and (3) poor quality of the harvested crop, either string beans or the threshed crop. Disfigured pods are unsalable as string beans, and apparently clean stock may develop the disease on the market or in transit to the market. Dry beans may suffer dockage on the market because of shrunken or spotted seed.

In many regions the disease takes a certain annual toll, and some years showing marked intensity or severity have been noted. Cases of individual fields showing losses of 100 per cent have been reported. Losses from the disease in Michigan were estimated to amount to $3,000,000 for the crop of 1915.

Etiology.—This disease is caused by *Colletotrichum lindemuthianum* (Sacc. and Magn.) Bri. and Cav., one of the imperfect fungi. The pathogen was first referred to the genus Gloeosporium, but the later discovery of setae in the acervuli led to the adoption of the present binomial, although some German writers still use the earlier name. Some claims of the occurrence of an ascus stage belonging to Glomerella have been made.

The mycelium, localized in the tissue of a lesion, organizes the fruiting bodies or *acervuli* which appear as stromatic layers from whose surface are formed closely packed, simple, erect, hyaline conidiophores, 45 to 55 μ in length. The conidia produced at the tips of the conidiophores are hyaline, continuous, oval or oblong, straight or slightly curved, 15 by 5 μ (13 by 4.44 to 22 by 5.33 μ) and accumulate to form pink, slimy masses. In some cases stiff, pointed, unbranched, septate, brown hairs

Fig. 155.—Semidiagrammatic section of pod lesion due to *Colletotrichum lindemuthianum*, showing relation of the anthracnose fungus to the tissues of the bean. (*After Whetzel, N. Y. (Cornell) Agr. Exp. Sta. Bul.* **255**, 1908.)

or *setae*, 30 to 90 μ long, arise between the conidiophores or around the margin of the acervulus.

The conidia from the acervular masses are separated during rain periods by solution of the cementing material and washed away or may be scattered to some extent by wind-blown or spattering raindrops. On germination on a susceptible host, a conidium produces a short germ tube which on coming in contact with the epidermis enlarges at the end and forms a brown, thick-walled, more or less angular cell, the *appressorium*. This is cemented to the cuticle and soon produces "a peglike infection hypha" which penetrates the underlying cell walls; this gives rise to branches which spread through the tissue and a typical lesion soon results.

One of the important features of the disease is the penetration of the pathogen into the seed where it may pass the winter as a dormant mycelium. When infected seed start germination, the fungus resumes activity and soon becomes spore-producing, thus constituting the most

important source of the first infections which appear in the fields. After the initial infections, new sources of spores become available and the disease will spread from plant to plant through the field. Any processes which transfer moisture from one part of a plant to another, such as splashing raindrops, dripping or running of moisture, or agitation by pickers of vines when wet with rain or dew, may carry the spores to new locations and thus spread the disease.

Free conidia are not viable to any extent after seven weeks, hence cannot carry the fungus over the winter. The fungus is, however, able to live over the winter to some extent on old affected vines and pods, but infections from such sources appear of minor consequence. Infected seed constitute the principal source of new infections.

Conditions Favoring Anthracnose.—The two important factors influencing the severity of the disease are temperature and moisture. The optimum temperature for the growth of the fungus ranges from 71.6 to 73.4°F., with a maximum of 86 to 93°F. New infections will form between 57 to 80°F. In the eastern United States the average climatic conditions are generally favorable for anthracnose, and epiphytotics can largely be explained by the proper combination of favorable temperatures with increased rainfall through a period of years. In the South (Louisiana), the disease is severe in the first part of the growing season but is absent in June or August since the temperatures are close to or beyond the maximum endured by the pathogen, but, in New York, temperature and moisture conditions may cause late plantings to suffer heavy damage. Low night temperatures, scanty rainfall, meager dews and an abundance of sunshine are factors which have excluded the disease from most of the area west of the Rocky Mountains. Because of the moisture relation, check-row planting gives less of the disease than continuous drills.

Host Relations.—Anthracnose is primarily a disease of various varieties of common beans (*Phaseolus vulgaris* L.), but it has been reported on Scarlet and White Runner, Lima beans, tepary bean, cow peas, common peas, horse beans and jack beans. On none of these, however, is it sufficiently severe to become epiphytotic.

There are numerous reports indicating varying degrees of susceptibility of the common bean varieties to anthracnose, but recent investigations, which have definitely established the occurrence of physiological strains of the pathogen, have rendered many of these older reports valueless. The following is a brief tabulation of the reports concerning physiologic strains:

Alpha and beta strains (Barrus, 1918)
Gamma strain (Burkholder, 1923)
8 distinct strains (Leach, 1923)

4 strains, distinct from American (Muller, Holland, 1926)
9 strains, distinct from American except one (Budde, Germany, 1928)
12 strains (Peuser, Germany, 1931)
34 strains in three groups, each of which contains one of the American strains of
Barrus and Burkholder (Schreiber, Germany, 1932)

Some progress has been made in the production of anthracnose-resistant varieties, by crossing and selection, but success is complicated by the number of physiologic strains. Mention may be made of a resistant White Marrow from crossing White Marrow and Wells' Red Kidney (Burkholder); a pea bean by using Michigan Robust as a susceptible parent (McRostie); crosses of White Imperial and Robust (Reddick); and, according to Schreiber, the Sugar Pearl was conspicuous for resistance, while pea bean No. 22 was immune to 30 of the strains and highly resistant to the other four.

Control.—First emphasis must be placed on the production and use of anthracnose-free seed. This can best be accomplished by the use of seed from localities in which the anthracnose does not occur. This is the basis for the important seed-bean industry of selected areas west of the Rocky Mountains, especially in Idaho and California.

Some of the practices that have been given attention are: (1) seed disinfection; (2) sorting by hand-picking or by flotation; (3) field selection for anthracnose-free pods; (4) avoidance of conditions favorable to infection; (5) early removal of affected seedlings and destruction of diseased vines in the fall; (6) spraying in the field or applying a fungicide to the picked pods before shipment; and (7) rotation to avoid residual contaminations. With seed disinfection shown to be ineffective, spraying of doubtful value from the standpoint of expense, and the various other practices of minor importance, the production and use of disease-free seed and the production and use of resistant varieties must be the refuge of the bean grower.

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DIPLODIA DISEASE OF CORN

Diplodia zeae (Schw.) Lev.

This disease has been called "mold," "moldy corn," "mildew," "rot," "ear rot" and "dry rot" because of the effects on the ears, but, since it also affects seedlings, the more general name of "Diplodia disease" is suggested.

The Diplodia disease is now recognized as causing one of the important ear rots of corn. It makes its maximum development in the corn belt

from Nebraska eastward but is known as far south as Florida and Texas and has been observed rarely in the Pacific Northwest. It has been studied in Australia, South Africa and South America.

Symptoms and Effects.—Three distinct phases are exhibited by the disease: (1) seedling blight; (2) crown infections; and (3) infections on maturing plants, the most important feature being a dry rot of the ears.

Fig. 156.—Normal ear of corn, and two ears affected with dry rot. (*From 22nd Ann. Rept. Neb. Agr. Exp. Sta.*)

When infected seed is planted, the stand may be reduced either by failure of grains to germinate or because seedlings are killed before they emerge from the soil. Some evidence has been presented to show that many seedlings from infected seed that do survive make weak plants which show an increase in nubbins and barren stalks over normal healthy plants, but it is believed that stand reduction and the resultant effect on yield are the important consequences from the planting of infected seed.

Infection from either soil or seed inoculum may cause *crown infections* which result in brown discoloration of crown and lower internodes, disintegration and shredding of internal tissues of crown, a brown discoloration of nodal plates, a dark brown decay of the mesocotyl with loss of

primary roots, and subepidermal pycnids may be formed on the crown and around the aerial adventitious roots (McNew, 1937).

The dry rot of the mature ears may show varying degrees of severity: (1) *light attacks* in which the kernels appear healthy from the surface but show a whitish mold covering the tips, or white flaky masses may be seen on the exposed-end surfaces when a cob is broken across; (2) *heavier infections* with mold very evident when the ear is husked; and (3) very *severe* infections with mold penetrating the husks and causing them to be discolored, being grayish or dirty looking or sometimes quite dark. In severe advanced stages, the ears may be erect rather than pendant, and be light in weight; the individual kernels dull in color, loosely attached and more brittle than normal; and minute black fruiting bodies may be found embedded in the flaky masses of mold. These black fruiting bodies are very conspicuous on the exposed surfaces when an ear is broken in two, thus constituting a distinguishing mark of the disease. Ears may sometimes be so lightly infected that the parasite is not in evidence until the seed is tested on the germinator.

Early infections may cause small shrunken ears of no value while less severe attacks produce ears of poor feeding quality. In severe attacks embryos are destroyed or viability is lost, while in more moderate attacks the viability is lowered or impaired, so that the corn is of no value for seed. The ear rot may continue to advance after the corn is picked, especially if the ears are soft and exposed to moisture.

The fungus may develop on various parts of maturing plants and cause reddish or purplish spots on the leaf sheaths, white wefts of mycelium on the shank of the ear or within the leaf sheaths, or large numbers of the fruiting bodies may appear at the shank and nodes, or in some cases on the stalks.

Dry rot causes (1) reduction in yield from poor stands, weakened plants and affected ears discarded at picking time; (2) a lowered market value of the harvested crop due to inclusion of partly molded ears; and (3) poor quality for seed purposes because of reduced germination and danger of seedling blight. Losses in Iowa in 1921–1922 were reported to range from 3 to 15 per cent of the ears at harvest, and caused an average damage to seed corn of 11 per cent. Losses in 1930 to 1933 in central Iowa ranged from 14 to 52 per cent.

Etiology.—Dry rot has been shown by pure-culture inoculations to be due to *Diplodia zeae* (Schw.) Lev., an imperfect fungus, which was first described as a saprophyte on old dead stalks. Another related species, *D. macrospora*, occurs in the southern United States and in South America, but is of minor importance. Infection is local, the principal point of entrance being the silks, tips of ears, ear shanks and nodes, the two latter being considered the chief points of attack. Infections take place after

flowering.   Although the fungus spreads from its point of entrance, it grows more rapidly in the languid tissues of the maturing plant parts and later continues a saprophytic life on the old stalks during the late fall or during the next season.

The fruiting bodies, described under Symptoms, are black, flattened spherical or pyriform, ostiolate *pycnidia* and produce many dark-brown, cylindrical to elliptical, obtuse, straight or slightly curved, one- (rarely two-) septate pycnospores, 24 to 33 by 5 to 52 μ.   The pycnospores are extruded in amorphous masses or in tendrils but readily separate in water.

Fig. 157.—Pycnid of *Diplodia zeae*, showing copious production of brown, one-septate spores.   (*From 22nd Ann. Rept. Neb. Agr. Exp. Sta.*)

The pycnidia may be immersed in the tissues of the stalks or ears or they may be free and seated in the dense masses of mycelium covering affected structures, especially in the ears.   They are uniloculate but sometimes lobulate with a common ostiole and produce the pycnospores from numerous, simple, hyaline conidiophores.   Fruiting bodies that resemble submerged pycnids have been produced in agar cultures and have been found in field samples from 14 states.   These may also be lobulated or cup-shaped and produce hyaline filamentous scolecospores, 21 by 45 μ, either alone or mingled with pycnospores.

The principal infection court is through the cavity of the leaf sheath, which becomes open by the loosening of the ligule soon after flowering. Masses of pollen, spores of the parasite, and moisture collect inside the leaf sheath and thus favorable conditions are afforded for germination of the spores which invade the tissues of the corn plant after first feeding on

the exosmosed sugar and the pollen. That infections can take place through the silks has been proved by artificial inoculations. Systemic infection has been suggested but not proved.

The pathogen can be carried over the winter on old stalks or discarded ears, since viable spores have been found in abundance on material one and two years old, and in traces even after three years. Mature spores germinate (see Fig. 158) at 10 to 15°C., but the optimum is 28 to 30°C.,

FIG. 158.—*A* and *B*, spores of Diplodia and their germination; *C*, normal starch grains in outline; *D*, partially digested starch grains from embryo of kernel affected by dry rot; *E*, partially digested starch grains from endosperm. (*From 22nd Ann. Rept. Neb. Agr. Exp. Sta.*)

and these temperatures are likely to prevail in the corn belt during the late summer when infections are known to occur. The disease may originate from spores produced by a previous crop on the same ground or from wind-blown spores, and it is possible that the pathogen may live for a time in the soil as a saprophyte. The mycelium produces enzymes by which it digests the constituents of the host tissue, especially starch grains and cellulose, and thus may greatly weaken affected stalks. It cannot contribute to the spoiling of ensilage, since it grows only under aerobic conditions.

A large number of physiologic strains have been recorded from various localities in the United States. In inoculation tests one strain exerts an inhibitory effect on others so that it finally predominates (Hoppe, 1936).

Conditions Favoring Dry Rot.—Heavy rains during the period when corn is approaching maturity (August and September) are very favorable for the development of the disease, since the temperature factor is also

genèrally close to the optimum for infection. Infection at this time is favored by: (1) the large amount of stored food; (2) the languid condition of the tissues; (3) the loosening of the leaf sheaths; and (4) the collection of water and spores between the leaf sheaths and the stalk.

Incipient infections may cause increased injury later in the season if the weather continues moist and warm, and dry rot may even continue to develop if the harvested crop is not sufficiently dried out or is exposed to rain by storage in open cribs.

Cool, moderately moist conditions following planting time favor seedling infections, while higher temperatures retard the progress of the disease, at least a larger number of seedlings emerge and grow to maturity resulting in better stands. Susceptibility to dry rot is increased by the bacterial blight and by second-brood chinch bugs. The severity of the disease is increased by chilling and freezing during the approach to maturity. No anatomical features are sufficient to account for the reaction of different strains of corn.

Control.—The facts presented under etiology suggest the following control practices: (1) *sanitary practices* including collection and destruction of infected ears at time of picking or husking, the destruction or removal of the stalks, and the avoidance of the use of contaminated barnyard fertilizer; (2) *crop rotation* to avoid residual soil contamination; (3) care in curing and storing the harvested crop to check progress of established infections; (4) the selection of seed corn from green standing stalks with sound shanks, with attention to careful drying and storage; and (5) the disinfection of the seed if taken from a crop known to be affected.

Disinfection of infected seed has been shown to increase the stand and lessen the amount of seedling blight. Increased yields have been reported from the use of mercuric dusts. Breeding offers some hope for the production of resistance. Some crosses developed in Rhodesia have given a flinty grain type that is resistant and equal to standard dent varieties in yield (Sansom, 1940).

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LATE BLIGHT OF CELERY

Septoria apii (Br. & Cav.) Chester and *S. apii-graveolentis* Dorogin

Celery is affected by two different fungous blights: (1) the late blight caused by two distinct species of Septoria; and (2) the early blight caused by *Cercospora apii* Fr. Following the first published report of late blight as a distinct disease of celery in Italy by Briosi and Cavara in 1890, it was recorded in 1891 from Massachusetts, New Jersey and Delaware and in the course of a few years was prevalent in almost all the celery-growing areas of the United States, including California (1897) and other western states. Nearly parallel with the development in America, the disease was noted in European countries including Denmark (1893), France (1894), Germany (1896), but not in the British Isles until 1910. In the earlier reports only a single species of Septoria was recorded as the causal agent, but in 1915 two distinct types of the disease were studied by Dorogin in Russia, the large-spot form, the common and generally recognized species, and the small-spot form, a new species. The former is reported to be the common form on celery in Europe, while the latter is more common and destructive in the United States. The occurrence of both species in the United States was established by the detailed study of Cochran in Michigan in 1932. It can now be stated that celery late blight is the most cosmopolitan and most serious of all celery diseases.

Symptoms and Effects.—The mature or completely developed lesions of the *large-spot type* are 1½ to 10 millimeters in diameter, "definite in outline, brown to reddish brown in color, and surrounded by a darker reddish brown border, with scattered pycnidia near the center." (Cochran). The lesions generally average 3 to 6 millimeters in diameter, and the black pycnidia are scattered singly in the center of the spot but are never clustered. Young infections are first in evidence as small chlorotic flecks which develop into definite lesions of dead brown tissue. The lesions generally begin on the older lower leaves and advance upward and when numerous may coalesce and cause the leaves to wither. Lesions on the leaf stalk are rare and of minor importance.

The mature or completely developed lesions of the small *spot type* are ½ to 3½ millimeters in diameter, "indefinite in outline, brownish to black or sometimes gray, with black pycnidia that may be found both in the necrotic portion of the spot and often in the green tissue surrounding it." The cells in the center of the spot are killed and the lesion advances outward. The pycnidia become more evident as black erum-

pent bodies "closely crowded together and often fused together in twos and threes." In the small-spot type the leaf stalks are also attacked, and the lesions may be scattered or if numerous may converge and involve

Fig. 159.—Leaf showing severe infection of the large-spot form of the blight of celery. (*After Foster and Webber.*)

extensive areas. In such severe infections the market product is poor or almost valueless from the blight alone or from the inroads of accompanying rot-producing fungi.

The above descriptions have emphasized the differences in the two forms: (1) the size and outline of the lesions; and (2) the time of formation and distribution of the fruiting bodies.

Etiology.—The large-spot type of late blight is caused by *Septoria apii* (Br. & Cav.) Chester and the small-spot type by *S. apii-graveolentis* Dorogin. It has been demonstrated by pure cultures and inoculations that the two species are distinct, but no perfect or ascus stages have been found in nature and all attempts to produce one in cultures have failed.

The mycelia of the two species are composed of irregularly septate, interwoven, dark hyphae and at intercellular corners may be grouped in fascicles or knots of irregular form. The hyphae of the small spot range in diameter from 1.5 to 4.5 μ and may extend some distance beyond

Fig. 160.—Section of a pycnidium showing spores oozing through the ostiole. (*After Cal. Bul.* **208**, *C. N. Jensen drawing.*)

the edge of the collapsed tissue, while in the large-spot, hyphae are 1.0 to 5.5 μ in diameter and may be absent in portions indicating the toxic action of the parasite.

The pycnidia of the large-spot form when present are confined to the central portion of the lesion and are spherical, 65 to 95 μ in diameter, and are borne singly except in old lesions that have been kept very moist. The spores are 13.5 to 34.2 μ by 1.0 to $2\frac{1}{2}$ μ, straight or slightly curved, and continuous to four-septate. The pycnidia of the small-spot form appear very early before the lesions are definitely outlined, range in size from 73 to 147 μ, and are usually crowded or even fused in groups. The spores are 22.5 to 58.5 μ by 1.5 to 3 μ, slightly flexuose, zero to seven septate, blunt at the ends and contents more or less granulose. During warm rains the spores are extruded and washed away, but with the cessation of the rain they may accumulate as cirri or spore masses at

the mouth or ostiole of the pycnids and are washed away by the next rain.

While rains and heavy fogs play a prominent part in the dissemination of the spores, it has been pointed out that irrigation water when allowed to flow along the furrow or trench in which the plants are set is a very important agent in spore transport. This has been demonstrated by the light occurrence of late blights when the irrigation trenches are between the rows as contrasted to the heavy infection when the plants are set in the irrigation trenches. It seems probable that wind and insects are of minor importance in spore dissemination but that some spread may result from the movement of tools, horses and men in the cultural operations.

Since no ascus stage is produced, the blight fungi must live over winter in the form of either pycnidia or hyphal masses in the refuse of diseased plants left in the field after harvest, or mingled with compost which may be spread upon the field or added to the seed beds in which seedlings are grown. In addition the mycelium may be found in walls of the fruits (seeds) and pycnidia may be developed and thus furnish inoculum for the spread of the blight. The spores formed in the old leaves that are partly decayed may remain viable and cause infections the next season, but not after a longer interval. The pycnidia on the seed and pedicels are more important as a source of viable spores, but most workers report that they are dead after two to three years. Thus new seed if bearing pycnids may spread infection, while aged seed may be used with little danger of starting the blight.

The effect of different temperatures on the germination of the spores and the development of the mycelium in cultures has been studied in some detail by Cochran. The large-spot form showed a low percentage of germination, 26 to 40 at the most optimum temperatures, while the small-spot species showed a much higher percentage of germination, or 88 to 94 through a wider range of temperatures. The effect of temperature on the size of the colonies on potato-dextrose agar showed the best growth of the small-spot form at 17 to 23°C. and the best for the large-spot species at 22 to 25°C. The inhibition of the growth of the small-spot form at low temperatures suggests the possibility of preventing damage in storage by reducing the storage temperature.

The early studies with the large-spot form showed that the hyphae enter the leaf by growing through the epidermal walls and then develop in the intercellular spaces and cause the characteristic spots. Later studies of both species have shown that the infection hyphae may enter through either the upper or lower epidermis but that no stimulus attracts the germ tubes to stomata, since hyphae may pass over stomatal openings without entering.

Varietal Relations.—The early reports showed that the Golden Self Blanching, a popular and profitable variety, was very susceptible. In general other easy blanching varieties were easily infected and in experimental tests lost their color and died, while the green varieties were able to endure the effect of both Septoria species for a longer time. Varieties of celery and celeriac are the only susceptible hosts, all other species of Umbelliferae being immune, on the basis of inoculations of over 60 different species.

Control.—No single practice can be specified which will control late blight, but attention should be given to the following:

1. *Sanitary Measures.*—*a.* In the field, trimmings from diseased plants should be removed and destroyed rather than scattered on the ground. Single plants that are very heavily infested should be removed rather than trimmed. In preparing celery for market in the crating or packing shed infected leaves will be removed, but these should not be thrown on the compost heap from which the pathogen may be returned to the field.

b. Avoid sites with poor drainage, or provide adequate drainage to reduce the soil moisture. Close planting and double rows increase the trouble in contrast to well-spaced plants in single rows.

c. Do not carry out harvesting operations when the plants are wet with dew or rain.

2. *Cultural Practices.*—*a.* Blanching by the use of boards is less favorable to the disease than dirt blanching.

b. Irrigating by running the trenches between the rows reduces the disease in contrast to setting the plants in the irrigation trenches, since in the latter the spores are spread from plant to plant by the water flow.

c. Give consideration to the use of fertilizers or stimulants. Fertilizers like nitrate of soda, sheep manure or a complete fertilizer which increase the succulence and the rate of growth favor infection, while the disease is reduced by applications to the soil of calcium sulphate, hydrated lime, and by weak solutions of either lithium nitrate or chloride.

d. Rotation with other crops so as to grow celery not oftener than once in three years.

3. *Seed Treatment.*—Since the fungus may be carried on or in the seed it is of importance to eliminate this source. This may be done in two ways: (*a*) by the selection of seed from fields or localities free from the disease; and (*b*) by the use of old or aged seed (over two years old), as the fungus in such seed will have died out completely or show a very low viability. If neither of the above specifications can be fulfilled some fungicidal treatment should be carried out. The following have been used and recommended by various investigators: (*a*) hot water, 30 minutes at 118°F., followed by a cold rinse and drying; (*b*) formalde-

hyde, 1 to 400–600 for 3 hours; (c) corrosive sublimate, 1 to 1000 for 15 minutes; (d) hydrogen peroxide for 3 hours (English workers). In addition Uspulun, tillantin and copper sulphate have been used.

4. *Spraying or Dusting.*—One or two applications of the selected fungicide should be made to plants in the seedbed, followed by later applications at week to ten-day intervals after plants have been set in the field, to be continued until seven to ten days before harvest. The following have been recommended: (a) Bordeaux, 4–4–50; (b) Burgundy mixture, 4–5–40; (c) monohydrated copper sulphate—lime dust, of varying proportions, such as 15–85, 20–80 or 25–75; and (d) cuprocide at the rate of 50 pounds per acre, or reduced to 25 pounds for the second and third applications. It has been pointed out that dusting in the morning when the leaves are wet with dew gives better control than applications made at midday or in the evening. Bordeaux has given best results as a spray.

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IMPORTANT DISEASES DUE TO IMPERFECT FUNGI

For key references to these diseases see F. D. Heald, "Manual of Plant Diseases," 2d ed., pp. 701–711.

Principal host	Common name of disease	Scientific name of causal organism
Pineapple	Blackheart or rot	*Thielaviopsis paradoxa* (d. Seyn.) v. Höhn.
Potato	Skin spot	*Oöspora pustulans* O. and W.
Lettuce and numerous other hosts	Black root and leaf blight or gray mold	*Botrytis cinerea* Pers.
Cotton, etc	Texas root rot	*Phymatotrichum omnivorum* (Shear) Duggar
Carnation	Bud rot	*Sporotrichum anthophilum* Peck.
Potato, etc	Wilt	*Verticillium alboatrum* R. and Ber.
Apple and pear	Blue mold	*Penicillium expansum* Lk.
Tomato	Leaf mold	*Cladosporium fulvum* Cke.
Peach	Freckle or scab	*C. carpophilum* Thüm.
Barley	Stripe disease	*Helminthosporium gramineum* (R.) Erick.
Barley	Net blotch	*H. teres* Sacc.
Barley	Spot blotch	*H. sativum* P. K. and B.
Carnation	Leaf mold, ring spot or fairy ring	*Heterosporium echinulatum* (Berk.) Cke.
Potato	Silver scurf	*Spondylocladium atrovirens* Harz
Rice	Blast	*Piricularia oryzae* Cav.
Beet, etc	Leaf spot	*Cercospora beticola* Sacc.
Chestnut and oak	Strumella disease	*Strumella coryneoidea* Sacc. and Wint.
Cabbage	Wilt or yellows	*Fusarium conglutinans* Woll.
Flax	Wilt	*F. lini* Bolley
Tomato	Wilt	*F. lycopersici* Sacc.
Potato	Wilt or blight	*Fusarium oxysporum* Schlecht
Cotton	Wilt	*F. vasinfectum* Atk.

MELANCONIALES

Grape	Anthracnose	*Gloeosporium ampelophagum* Sacc.
Citrus	Scab	*G. fawcettii* (Jenkins)
Apple	Superficial bark canker	*Myxosporium corticolum* Edg.
Cucurbits	Anthracnose	*Colletotrichum lagenarium* (Pass.) E. and H.
Citrus spp	Wither tip	*C. gloeosporioides* Penz.
Lettuce	Ring spot	*Marssonina panattoniana* (Berl.) Magn.
Stone fruits	Blight	*Coryneum beijerinckii* Oud.
Cherry and plum	Leaf spots	*Cylindrosporium spp.*

SPHAEROPSIDALES

Apple	Blotch	*Phyllosticta solitaria* E. and E.
Apple	Fruit spot	*Phoma pomi* Pass.
Crucifers	Blackleg	*Phoma lingam* (Tode) Desm.
Tomato	Fruit rot	*P. destructiva* Plow.
Eggplant	Blight	*Phomopsis vexans* (Sacc. and Syd.) Hart.
Citrus fruits	Melanose and stem-end rot	*P. citri* Faw.
Cranberry	End rot	*Fusicoccum putrefaciens* Shear
Poplar and willow	Canker	*Cytospora chrysosperma* (Pers.) Fr.
Citrus	Diplodia disease	*Diplodia natalensis* Evans
Citrus	Knot	*Sphaeropsis tumefaciens* Hedges
Chrysanthemum	Ray blight	*Ascochyta chrysanthemi* Stev.
Clematis	Stem rot and leaf spot	*A. clematidina* (Thüm.) Gloy.
Gladiolus	Heart-rot disease	*Septoria gladioli* Passer
Tomato	Leaf spot	*S. lycopersici* Speg.
Poplar	European canker	*Dothichiza populea* S. and B.

CHAPTER XIII

BACTERIAL DISEASES OF PLANTS

Previous to 1878, the occurrence of bacterial diseases of plants had not been demonstrated, although bacteria had already been accepted as important pathogens for man and domestic animals. Our knowledge of bacterial diseases of plants is, therefore, the result of studies made within the last 50 years, but the most rapid advancement has been made during the last 25 years.

Fig. 161.—Diagram illustrating the morphology of the true bacteria. A, coccus forms; B, bacillus forms; C, spirillum forms; D, types of spore formation.

Since the demonstration of the bacterial origin of fire blight of pear and apple by Burrill in 1878–1883, a large number of bacterial diseases of plants have been described. In 1920 Smith reported specific bacterial diseases on hosts scattered through more than 150 genera in over 60 families. Elliott in her Manual of Bacterial Pathogenes published in 1930 listed 177 species of bacteria pathogenic on plants.

General Morphology of the Bacteria.—The plant body in its simplest form in the true bacteria (Eubacteriales), sometimes called the *lower bacteria*, consists of a single cell, which can be assigned to one of three general types: (1) globular or spherical forms, the *coccus type;* (2) short to long cylindrical or rod shaped, the *bacillus type;* and (3) the short or long, spiral, cylindrical forms, the *spirillum type*. The cells of the different types may be held together as pairs, chains, long or short filaments,

long spirals or, in the coccus forms, as packets or cell masses, but, in every case, the single cell is to be considered as the individual and the various groupings, as aggregates of individuals. Under certain conditions, irregular or branched forms may be assumed. The bacteria vary in size from coccus forms 0.15 to 1 μ in diameter to bacillus or spirillum forms 0.3 to 3 μ in diameter and 1 to 6 or more microns in length.

Each cell is surrounded by a definite cell wall or membrane, which is nitrogenous in chemical character. This wall may undergo a mucilaginous modification causing viscid or slimy groups held together as sheathed filaments, irregular masses or thin, scumlike sheets. Certain species of

Fig. 162.—Poured-plate isolation showing colonies of bacteria obtained from a bacterial blight of Antwerp raspberries.

bacteria are always inactive or incapable of locomotion, while others under certain conditions are able to swim actively through a liquid medium, or have the power of locomotion. This motility is the result of the lashing movements of delicate vibratile threads, the *flagella*, varying in number and distribution.

Reproduction is by simple fission or cell division, hence the common name, the *fission fungi*. Under favorable conditions, the separation of a cell into two may be completed in 20 to 30 minutes. Some species of bacteria are able to form *endospores*, by the contraction and concentration of the protoplasmic contents into a globular or oval body lying within the parent cell, but surrounded by a firm solid membrane (Fig. 161.). These endospores are very resistant to desiccation, heat or other unfavor-

able factors and, under suitable conditions, may germinate to form vegetative cells. None of our plant pathogens are known to produce spores.

Classification of Bacteria in General and of Bacterial Plant Pathogens.—According to the recent classification by the Committee of the Society of American Bacteriologists (Bergey's Manual, 1923, 1930), the Schizomycetes or bacteria are arranged in the following six orders:

Order J.   Eubacteriales, the true bacteria
Order II. Actinomycetales, the mold-like bacteria
Order III. Chlamydobacteriales, the alga-like iron bacteria
Order IV. Thiobacteriales, the alga-like sulphur bacteria
Order V.   Myxobacteriales, the myxobacteria
Order VI. Spirochaetales, the protozoan-like bacteria or Spirochaetes

The forms that are of phytopathological importance belong to the family of the Bacteriaceae of the true bacteria. Plant pathologists generally agree that the Actinomycetales are imperfect fungi rather than bacteria, consequently *Actinomyces scabies*, the cause of common scab of the Irish potato, has been considered in the chapter on Diseases Due to Imperfect Fungi.

FIG. 163.—Comparative size of apple-scab spore and three species of bacteria. *A*, crown-gall bacteria; *B*, bacteria of black rot of cabbage; *C*, fire-blight bacteria.

All of the bacterial plant pathogens belong to the rod forms and are grouped into three genera, Bacterium, Pseudomonas and Bacillus by Migula; and Aplanobacter, Bacterium and Bacillus by Smith; while they are placed in two genera, Phytomonas and Erwinia by the Committee of the Society of American Bacteriologists.

This classification is presented in the following tabular comparison:

Nonmotile	Motile	Motile
Bacterium: Migula Aplanobacter: Smith	Pseudomonas: Migula Bacterium: Smith	Bacillus: Migula Bacillus: Smith
Phytomonas: S. A. B.		Erwinia: S. A. B.

Types of Bacterial Diseases.—Three types of bacterial disease may be recognized:

1. *Vascular diseases* are characterized by primary invasions of the water-conducting vessels of the fibrovascular bundles by the bacterial pathogen. In diseases of this type the water-conducting vessels may become so filled with the bacteria that water can no longer be supplied to the foliage, and the plant wilts rather suddenly. This is well illustrated in the wilt (*Bacillus tracheiphilus*) of cucurbits, the brown rot (*Pseudomonas solanacearum*) of the potato, eggplant, tomato, tobacco and other species of the nightshade family, and by the black rot (*P. campestris*) of the cabbage, cauliflower and other members of the mustard family.

2. *Parenchyma diseases* are troubles in which the pathogen invades the soft or succulent parenchyma tissues of the host, as a primary fea-

Fig. 164.—Bacterial leaf spot of the common barberry.

ture, but may sometimes break into adjacent vascular elements. In such case there is a necrosis of tissue without hyperplasia, and a spotting, blighting or rotting of the affected parts is the final result. This type is well illustrated by fire blight (*Bacillus amylovorus*) of apples and pears, the soft rot (*B. carotovorus*) of carrots and many other fleshy roots, stems, rhizomes or fruits, the blackleg (*B. atrosepticus*) of the potato and the angular leaf spot (*Pseudomonas malvacearum*) of cotton.

3. *Hyperplastic diseases* are characterized by tubercle, tumor or gall formation or by the development of additional organs (shoots or roots) from adventitious, dormant or latent buds. In diseases of this type, the bacteria stimulate certain cells to increased activity, and, as a result of this abnormal cell division, the structures noted above are developed. Notable illustrations of this type are the well-known crown gall (*Pseudomonas tumefaciens*) of fruit trees, shade trees and many other hosts, the hairy root (*P. rhizogenes*) of apple and other trees causing an excessive number of small fibrous roots and the olive knot or tubercle (*P. savastanoi*) causing irregular aerial knots or excrescences.

How Bacteria Invade Their Hosts.—The external surface of the plant body of seed plants is covered in large part by an epidermis with an external cuticle or also with cuticularized external walls, or by a more impervious layer of cork cells, the periderm. Some parasitic fungi are able to make their way through such unbroken epidermal walls, but pathogenic bacteria seem unable to penetrate cuticularized walls or layers of cork cells. This leaves wounds, natural openings or surfaces unprotected by an external cuticle as the possible avenues of entrance.

1. *Entrance through Wounds.*—Mechanical injuries of various kinds, which bruise or break the tissue, subject the injured organs to invasion by wound parasites. The cell sap and the protoplasmic contents from

FIG. 165.—*A*, portion of a leaf section showing bacteria in water film above a stoma; *B*, penetration of the bacteria into the substomatal chamber; *C*, bacteria in the intercellular spaces of a parenchyma tissue; *D*, bacteria advancing in a spiral vessel. All semidiagrammatic.

the injured cells offer a food or culture medium in which bacteria may be lodged and in which they can make their initial development. By repeated fission, a mass consisting of many bacteria may result and adjacent uninjured cells may be affected as the bacteria work their way into the deeper tissues. Notable examples are: olive knot and fire blight through hail wounds; crown gall through budding or grafting wounds; soft rot through digging injuries in potatoes, carrots and other root crops; wilt of solanaceous plants through nematode injuries; wilt of cucurbits through feeding of cucumber beetles, fire blight through tunnels or punctures by various insects, and cereal-leaf infections through guttation ruptures.

2. *Entrance through Stomata.*—The way in which stomatal invasion may take place is illustrated in Fig. 165. Bacteria located in a film of

water over a stomatal opening divide in rapid succession, and soon work their way into the stomatal opening and the substomatal chamber from which they push on into adjacent intercellular spaces. Since the first proof of stomatal invasion in black spot (*Pseudomonas pruni*) of the plum, numerous other cases have been studied.

3. *Entrance through Water Pores.*—The most noteworthy case of water-pore invasion is to be found in the black rot of the cabbage. The bacteria develop first in the tissue immediately below the water pores but soon make their way into the spiral vessels of the vein terminal and then advance down the vessels as cell division proceeds, rapidly producing the characteristic black venation.

4. *Entrance through Nectaries.*—The nectar-secreting glands of flowers offer another possible place of entrance, since the glandular tissue has no protection of cuticle or cuticularized layers, and the sweetish nectar presents a food that is favorable for the growth of bacteria. The blossom blight phase of fire blight (*Bacillus amylovorus*) of apple and pear offers the most common case of entrance of the pathogen through the nectaries. There are but few other proved cases of nectrial infection.

5. *Entrance through Lenticels.*—The extent to which bacteria can enter through these passageways which lead through the corky bark into the underlying parenchyma tissue of twigs, woody branches or modified stems is uncertain. This seems to be a possible way of entrance of bacteria into potato tubers, especially those which cause soft rot. The possibility of lenticellate infection is one worthy of more detailed investigation.

The Location of Bacteria in Diseased Tissue.—In a few cases bacterial diseases are caused by the development of the pathogen between certain closely appressed organs, for example, between the glumes of the affected heads in Rathay's disease of orchard grass and between the outer petals of unopened buds in the gum-bud disease of carnations.

Bacteria which actually penetrate the tissues may be: (1) *intercellular*, or in the spaces between cells, as in most of the parenchyma diseases; (2) *intravascular*, or in the water-conducting vessels of the xylem, as in the wilts and other vascular diseases; and (3) *intracellular*, or within the interior of cells. In invasions which are primarily intercellular or during the first stages of intercellular infection, the bacteria may enter the vessels to a limited extent. In many vascular invasions, the bacteria later break out into the surrounding parenchyma tissue and form intercellular pockets, while in advanced decompositions of tissue, bacteria may enter the dead cells and assist in the work of destruction. In a few cases, however, the bacteria gain access to the interior of living cells at the very beginning. This would seem to imply the ability of the pathogen to penetrate unbroken walls of cells by its own activities, possibly by digestive action and perhaps by entering minute wall perforations.

The Action of Bacteria on Their Hosts.—Owing to rapid multiplication in the intercellular spaces, bacteria may cause mechanical splitting, tearing or crushing of tissues, but their principal action is chemical including: (1) the *separation of cells* from each other by the digestive action of enzymes upon the pectic substances of the middle lamellae, leading to the production of cavities in various tissues; (2) the production of enzymes which convert starches into sugars, complex sugars into simpler forms or digest and make possible the assimilation of proteids and other nitrogenous compounds; (3) the formation of injurious acids, alkalies or toxic substances of some other character as by-products of their activity; and (4) the production of substances which stimulate cells to abnormal activity.

The Reaction of the Host.—If it were not for host resistance, parasites when once established would always multiply indefinitely until a fatal ending would curtail their activities, but we know that many bacterial lesions are of limited extent and that finally the intruding organisms die out and disappear, leaving but the marks of the battle, while the host still lives.

In addition to the killing effect upon host tissues, some of the more striking effects are: *retarded or lessened growth* of certain groups of tissues, certain parts or organs or the entire plant; *changes of color*, involving conservation of chlorophyll, with the development of a deeper green or the change to yellow or some other color; *distortions* of leaves, stems or other parts not due to hypertrophies or to hyperplastic changes; and, finally, the *development of new tissue*, new organs or abnormal overgrowths as a result of either *hyperplasia*, or increased cell division, or of the hypertrophy of cells.

In some of the simple bacterial attacks, the advance of the bacteria in the tissues may be checked by the construction of a barrier of cork cells in advance of the invaded tissues. In other cases the increased cell activity, expressed in hyperplasia, may be so directed as to cause the development of organs of normal structure, roots or shoots, but either out of place or in excessive numbers (hairy root and witches'-brooms). In other cases of hyperplastic response, cell division runs riot, unchecked and undirected, with the result that irregular or formless overgrowths, cankers, tubercles, tumors or galls are formed. In these cases it is probable that some chemical substance produced by the bacteria or by the irritated cells excites the cells to cell division, which proceeds with great rapidity.

The Dissemination of Bacterial Diseases.—Some of the more important agents of transmission may be mentioned: (1) *seed* used in the broadest sense to include true seeds, fruits, bulbs, tubers or other propagating stock; (2) *insects and other animal life,* including birds, mollusks and

worms; and (3) *contaminated fertilizer* from a compost heap may harbor certain pathogens.

BLACK ROT OF CRUCIFERS

Pseudomonas campestris (Pam.) E. F. S.

The black rot of the cabbage and other crucifers is a vascular bacterial disease which has been referred to as a "bacteriosis," "bacterial rot," "brown rot," or, most frequently, as the "black rot."

The true nature of this disease was first demonstrated by Pammel in 1893–1895 in a study of a bacteriosis of rutabagas in Iowa. During the next few years, the disease was studied intensively on cabbage and other crucifers, especially by Smith of the U. S. Department of Agriculture and various workers in Wisconsin, New York and, later, by European workers and other American investigators.

The disease has been reported from practically all of the states east of the Mississippi river and from several west of it but is rare west of the Rockies. For twenty-five years or more, it has been a destructive disease in the older, cabbage-growing sections of the country. It is well known in Europe, and has been found also in the West Indies, New Zealand, South Africa and the Philippines.

Symptoms and Effects.—The earliest symptom of the disease, especially in the cabbage, is generally a yellowing of the foliage with a blackening of the veins, beginning at the leaf margin or around some insect injury, with a progressive development of the stain downward into the petiole and then into the vascular elements of the main stem. This invasion causes the leaves to wilt, turn yellow and dry up, and there may be a gradual shedding of the lower leaves, finally resulting in a long bare stem marked only by leaf scars, and small axillary shoots, with distorted leaves at the crown. Early attacks may destroy the young plants even before the unfolding of the first leaf, and later attacks cause dwarfing and a crippled development without the formation of a head.

A blackening of the vascular bundles may be noted in cross sections of the petioles of invaded leaves, and, in well-advanced cases, the wood cylinder of the stem will show a brown or black color. In affected turnips a yellowish slime may ooze out from the cut ends of vascular bundles, while in some succulent structures pronounced cavities may be formed.

In the cauliflower, infections occur over the entire leaf, instead of along the leaf margins, causing leaf perforations in dry weather, or they may spread and cause a wet stump rot under more humid conditions. Numerous leaf-spot infections are also characteristic of the trouble on Chinese cabbage.

Plants affected by the pathogen alone have no pronounced odor, but other bacteria may enter and cause a destructive soft rot, transform-

ing a head into a black, slimy, foul-smelling mass. Field losses of 40 to 50 per cent have not been uncommon (even 90 to 100 per cent) and rotting may be continued into storage.

Etiology.—This disease is caused by *Pseudomonas campestris* (Pam.) E. F. S. which was originally described as *Bacillus campestris*. The pathogenicity of this bacterium was first proved for rutabagas and turnips and later for cabbage, cauliflower, kale, rape, radish and black mustard, by the use of pure-culture inoculations. The rods are 0.4 to 0.5 by 0.7 to 3 μ and occur singly or in long chains. When crowded in the plant or in old cultures, the rods are very short, almost coccuslike, but, in

Fig. 166.—Cabbage leaf showing manner of infection. *A*, holes eaten by insects; *B*, diseased areas unshaded except blackened mesh of veinlets; *C*, blackened veinlets affected by disease; *D*, water pores through which the black-rot bacteria enter to produce a marginal infection. (*After Russell, Wis. Bul.* **68.**)

young infections or cultures, they are much longer than broad. Young bacteria are actively motile by means of a single polar flagellum. No endospores are formed.

While wounds caused by mechanical injuries and leaf-feeding insects may be the avenue of entrance for the pathogen, the majority of the infections take place through the normal leaf openings, the water pores or hydathodes of the leaf margins, or in some hosts by stomatal entrance. During cool, moist nights, liquid moisture exudes from these water pores, and, if the pathogen reaches this moisture and atmospheric conditions delay evaporation, the pathogen may multiply, enter the hydathode chamber and soon reach the adjacent vascular elements of the veins. After this, the characteristic leaf symptoms will follow with some rapidity. Invasion through ordinary stomata has been demonstrated on

cabbage cotyledons and is probably true for the leaf-spot type of the disease in cauliflower and Chinese cabbage.

The bacteria multiply in the spiral and other vessels, move gradually forward as they increase in numbers, finally reach the base of the leaf and enter the vessels of the main axis. From this point, they may be distributed to other leaves, the movement outward or upward being aided by the transpiration stream and the mobility of the rods. After the organism has penetrated the central axis of the host, wilting of leaves may result from the plugging of the vessels in the petioles by bacterial

Fig. 167.—Section of a cabbage leaf parallel to the surface near the margin, showing the result of infection by black-rot bacteria through the water pores. (*After E. F. Smith.*)

aggregates, even before a general penetration has taken place. The bacteria also break out from the fibrovascular vessels into the intercellular spaces of surrounding parenchyma. Their passage between such cells is facilitated by the secretion of enzymes which dissolve the middle lamellae. The lignified elements are not digested but the nonlignified cells may be entered and destroyed. The destruction of parenchyma tissue is the cause of cavities noted in the description of Symptoms and Effects.

In any occurrence of the disease the source of the primary infections may be from: (1) bacteria carried on the surface of the seed; (2) from manure or discarded refuse containing the pathogen; (3) from bacteria

living over in the soil from a previous diseased crop; (4) the disease may be carried to the field by seedlings from a contaminated seedbed; or (5) volunteer plants may be infected and tide the organism over the summer period. After the disease is established in the field, it may be spread by natural agencies such as insects, rain water or irrigation water from the infected plants to healthy plants. Seed of cabbage and some other crucifers is contaminated during threshing, but, in the cauliflower, the bacteria may penetrate the seed pods and seed coats previous to harvest.

Host Relations.—The disease is known to infect cabbage, cauliflower, brussels sprouts, rape, collards, kale, rutabagas, turnips, radishes and mustard. It has been found on winter stock (*Matthiola incana*) in Germany and recently on Chinese cabbage (*Brassica chinensis*). Radishes are rather resistant, turnips and rutabagas more susceptible and cauliflower very susceptible. The Houser cabbage has been reported as "practically immune to black rot under field conditions." It should be noted, however, that the yellows-resistant varieties developed in Wisconsin have not proved resistant to black rot "or to the other common cabbage diseases such as blackleg (Phoma) and clubroot (Plasmodiophora)."

Prevention or Control.—The following are control practices of value:

1. The use of seed from regions known to be free from this disease. The seed from the fields of Mt. Vernon, Washington and adjacent territory is notable in this respect.

2. Seed disinfection unless the seed is known to be free from contamination. Steeping in mercuric chloride (1–1000) for 30 minutes, followed by a cold-water rinse and then drying is effective for cabbage, but it does not prevent the disease on cauliflower because of intraseminal infection. For this reason and because the treatment is not effective against blackleg, the hot-water treatment is recommended: cabbage and brussels sprouts, 25 minutes and cauliflower 18 minutes at 122°F., followed by a cold-water rinse and then drying. Because of probable injury a germination test should be made.

3. Disinfection of the seedbed with 1–1000 or 1–1200 mercuric chloride, by watering the soil. Three applications to young plants have given good control of the disease in cauliflower, and are also effective for clubroot and damping-off.

4. Cultural and sanitary practices including (*a*) late planting after July (especially for cauliflower); (*b*) avoidance of contaminated soil or manure for the seedbed; (*c*) crop rotation; (*d*) control of insect pests; and (*e*) the pulling and destruction of young plants as soon as detected.

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ANGULAR LEAF SPOT OF COTTON

Pseudomonas malvacearum E. F. S.

This disease of cotton is known in its various phases by a number of the common names such as black arm, vein blight, bacterial blight, bacterial boll rot and gummosis, but the more generally accepted name is angular leaf spot. It was first studied in Alabama by Atkinson in 1891 and since that time has been the subject of reports from workers throughout the cotton-growing areas of the United States and foreign countries. The disease occurs in either mild or severe form wherever cotton is a commercial crop, "ranging from a trace to as much as 40 to 60 per cent," and is most severe in the United States from the southeastern states to Arizona and southern California. The various species of Gossypium to which cotton belongs are susceptible to the disease. It has also been reported as causing spotting, defoliation and stunting of the Mexican variety "Pochote" of the Kapok tree (*Eriodendron anfractuosum*), and also occurs on *Thurberia thespesioides*, a wild cottonlike plant in Arizona and New Mexico.

Symptoms and Effects.—Four different common phases of the disease develop in accordance with the location of the seat of invasion: (a) leaf spots or other angular lesions on the leaves; (b) black lesions, the so-called *black arm* on the stems; (c) circular, black, slightly sunken lesions on the bolls causing *boll rot* and gummosis; and (d) seedling infections.

The common leaf lesions show first as small water-soaked spots which turn brown at the center and form angular dead areas with reddish border bounded by the veinlets. The individual lesions may be small or reach ¼ inch in diameter and may be well scattered or sometimes localized. Larger dead areas may be caused by the coalescing of numerous infections and lead to death and shedding of leaves. A less common type of leaf invasion is characterized by irregular marginal or internal lesions, 1 to 3 centimeters in diameter, in which the bacteria have been shown to advance along the veins rather than in the mesophyll.

In case infections occur on the stem, larger branches, twigs or petioles, elongated black lesions may be formed 1 inch or more in length. Under favorable conditions of temperature and moisture these lesions give off an ooze containing quantities of the pathogen. Severe infections may cause girdling and death of the affected organs, resulting in the condition

known as *black arm*.   The weakened or girdled branches may sometimes be broken by the wind.

The disease is first evident on the bolls as small, rounded, water-soaked spots which turn dark in the center.   These lesions may enlarge, and a few may cover the entire boll, and penetrate into the interior, or under less favorable conditions the lesions may remain small and not involve the central axis, while others located at the tips of the carpels

Fɪɢ. 168.—Angular leaf spot of cotton.   1, leaves showing two types of injury; 2, infections on squares and small bolls; 3, lesions on base of boll.   (*After Elliott.*)

may cause premature separation.   Primary boll lesions may be invaded by other species of bacteria and rot-producing fungi which unaided could not have penetrated, and the complex results in that phase of the disease called *boll rot* and *gummosis*.   Basal infections of flower buds and young bolls may cause these structures to be shed, thus increasing the damage.

Under certain conditions very young seedlings may develop infections in the stem and in seed leaves resulting in a condition resembling damping-off.   The infections may be localized and kill the tip of the seedling or all or only a part of the seed leaves and in the extreme infections cause the' death of the entire seedling.   Under certain conditions this phase of the disease may cause thin stands and reduced yields.

The extent of the reduction in yields from this disease in the United States is emphasized by the figures presented by the Plant Disease Survey of the Department of Agriculture: for 1920, a reduction of 213,000 bales; for 1939, a loss of 158,000 bales; and fluctuating losses for the intermediate years.

The disease reduces the yield of cotton by destroying a certain percentage of the chlorophyll-bearing tissue of leaves and stems and thus lowering the production of carbohydrates; by causing shedding of young bolls, rot of some that mature and by staining the lint or fiber; and by increasing breaking by wind.

Etiology.—Angular leaf spot of cotton is caused by *Pseudomonas malvacearum* E. F. S., a short, rod-shaped, one-flagellate bacterium, which produces circular, pale yellow colonies on nutrient agar. The rods are often joined in pairs or in short chains. The initial establishment of the pathogen in the field is on the young seedlings grown from contaminated seed, and under favorable conditions of moisture and temperature the other phases of the disease develop. It has been shown that under favorable conditions of moisture 20 per cent or more of the seedlings may develop the disease from internal contamination of the seed. Wounds are not necessary for infection, since the bacteria are able to enter through the stomata. Undoubtedly rain, especially when blown by wind, is the most important agent in the spread of the bacteria, but some dissemination may result from cultivation and from irrigation water. Insects are of minor importance, but may introduce the bacteria especially into the bolls.

Soil temperature and moisture are the most important factors affecting infection. Temperatures of 30 to 40°C. with a humidity of 85 per cent or more are very favorable, while lower temperatures and humidity retard infection and prolong the incubation period. It has been pointed out that primary infection is governed by the soil temperature at seeding time and during the first few days of germination and is highest at soil moisture approaching saturation. Secondary infections depend on the atmospheric moisture, which is especially important for 48 hours after inoculation, and the continuation of favorable temperatures during the incubation period of three to ten days. The bacteria on cottonseed have been shown to withstand 90°C. for 5 hours in dry air, or 1 hour in moist air. In pure culture they are killed in 20 minutes at 50°C. or in 10 minutes at 56°C. In pure culture they have endured freezing as low as −27°C. for one month, but were killed by alternating freezing and thawing up to 10.6°C. The bacteria in the leaves have remained viable for two months under snow, but were killed by alternate thawing and freezing.

Some workers have reported the most infection in the earliest sown fields and a decrease in later seedings. Rainfall periods are necessary for secondary spread, and the rate of spread depends on the frequency and severity of the rains. New fields may show the most infection on the side adjacent to old cotton land, the infective debris from an old crop being the cause of the outbreak.

Control.—Three different practices which have a bearing on control may be presented: (a) cultural operations; (b) seed treatments; and (c) selection and breeding of resistant varieties.

(a) *Cultural Operations.*—The incidence of the disease is increased by early planting as compared with late planting; by delayed thinning with the resultant crowding which offers better conditions for infections; by poor tillage; and by too late flooding in the case of irrigated fields. A lack of potash is reported to increase the severity of the disease. The various cultural operations may be used to reinforce the other control practices.

(b) *Seed Treatments.*—Chemical disinfection is generally recommended, the most widely used being the sulphuric acid delinting treatment, which is carried out by immersing the seed with frequent stirring in concentrated sulphuric acid for 5 to 15 minutes in earthen jars or wooden tubs coated with pitch and then rinsing thoroughly to remove the acid, after which the seed is thoroughly dried. A *second method,* the hydrochloric acid gas process has been developed by the United States Bureau of Plant Industry. This process which is feasible only on a commercial basis has the advantage that the seed is kept dry throughout the handling. A *third method* is the use of a dust disinfectant, some of the organic mercury preparations being most effective, when used at the rate of 4 ounces per bushel of seed. Thorough agitation for 15 to 20 minutes is sufficient, and such dusted seed may be planted at once or stored for later use. The importance of seed treatment in preventing seedling infection is so great that all plantings should be protected, as the disease may spread from untreated fields to adjacent fields free from the disease.

(c) Variations in variety resistance with breeding for the production of more resistant strains or varieties offers some hope. In tests on Upland cotton, using a grading from 0, or immunity to 12 as full susceptibility, variety resistance has been shown to range from 3 to 10. Complete immunity has been reported for some of the Old World species.

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FIRE BLIGHT

Bacillus amylovorus (Burr.) Trev.

This disease is frequently called "pear blight," but "fire blight" seems more appropriate since it expresses one of the symptoms characteristic of the disease on its various hosts.

Fire blight is a disease indigenous to North America where it probably occurred on wild hosts previous to the introduction of the cultivated varieties. It was first observed in Hudson River Highlands about 1780 and spread, westward and southward, until it became destructive in southern Canada and in the states of the northern Mississippi Valley. It extended through the South devastating the pear-growing districts of the gulf coast of Texas. Later it spread west of the Rocky Mountains to California, Oregon, Washington and British Columbia, but it has not been reported in Washington west of the Cascades, although present in the coast country both to the north and to the south. In addition to the North American continent the disease has been reported from Japan (1911), New Zealand (1919) and Italy (1924).

Symptoms and Effects.—According to the organs attacked various phases of the disease may be recognized:

1'. *Blossom Blight.*—Soon after the blossoms open they may turn brown and wilt without any set of fruit or in addition the discoloration may extend down the pedicels, until the adjacent leaves turn brown, wilt and remain hanging as a blighted tuft around a blasted group of flowers. If conditions are favorable, the discoloration may advance down into the fruit spur resulting in its death and may even continue into the branch at the base of the fruit spur. Pearllike droplets of bacterial exudate may appear on the pedicels if the atmosphere is relatively moist.

Fig. 169.—Blossom blight of Jonathan apple.

2. *Leaf Blight.*—Brown, more or less zonate lesions of varying shades of brown may appear on the leaf margins, leaf tip or sometimes internally, especially on pear and apple, and advance inward or downward. In some cases the advance of these lesions is checked and is never resumed, while in others the entire leaf blade is invaded, after which the blight may extend down the leaf stalk and cause a blighting of the twig. Pearllike droplets of ooze may be formed on the leaf lesions, or spread out as thin films which dry to form shiny flakes.

3. *Twig Blight.*—In this phase of the disease on the apple, a faint amber-yellow or reddish coloration shows at the tip, while, in the pear, the invaded structures are generally blackened. The bacteria may migrate from the twig into the leaf petioles or even into the leaf blade, but the leaves more frequently turn brown and dry out as an indirect effect of the twig lesion. The drooping terminal twigs with shriveled and curled drooping leaves, brown in the apple or black in the pear, which stand out in marked contrast to the normal green foliage, present a characteristic picture of twig blight. Viscid drops of exudate may appear on the parts as in blossom blight and in leaf invasions. The disease may advance downward through the twig and reach other branches, causing localized stem cankers, or an unchecked advance may continue into the body of the tree.

4. *Fruit Spot Blight.*—This may result from new infections on partially grown fruits. Pears and some of the more susceptible apples may be completely invaded, the pears blackened and the apples turned brown, while in the more resistant apples circular or slightly irregular,

localized, depressed, dark colored lesions may be formed. Drops of

Fig. 170.—Apple leaves showing natural leaf invasions by the fire-blight bacteria.

bacterial exudate are a frequent accompaniment of the complete fruit blight.

5. *Cankers, Body or Limb Blight or Collar Blight.*—When the disease invades limbs or main trunk of a susceptible host, *blight cankers* of limited size may result, or the infection may spread diffusely producing a *body or limb blight*, while a localization of the disease at the base of the main trunk may produce a *collar blight*. These several types may originate from primary infections through fruit spurs, normal twigs or water sprouts or from direct infection through wounds. When a bark lesion ceases to spread, the tissue dries out, shrinks somewhat and a slit or

Fig. 171.—Typical twig blight of Jonathan apple.

crack appears, separating the old dead tissue from the surrounding normal bark (see Fig. 173). These localized lesions of the canker type are

more common on apples than on pears.   The bacteria in a certain percentage of the lesions die out but, in others, remain alive at some point, and resume activity next season, thus constituting *holdover cankers*, which may serve as centers from which the bacteria may be spread to other limbs or adjacent trees.   Collar blight is indicated by dead, discolored and sunken bark at the crown, and trees affected with this form of the disease will show an unhealthy appearance of the foliage, especially in

Fig. 172.—Fire-blight lesions on green apples.

certain limbs.   The crown invasion may be completed in a single season in small trees or longer in large trees, but, when the trunk is completely girdled at the base, death results.   A somewhat similar collar injury may be caused by freezing or by some fungous root rots.

The injury from blight varies with the location, severity or number of lesions.   As a result of the disease the following injuries may result: (*a*) loss of foliage; (*b*) blighting of blossoms and, consequently, the failure to set fruit; (*c*) spotting or blighting of fruit that has escaped the ravages at blossoming time; (*d*) the dieback of twigs and branches from twig blight or limb cankers which girdle the axis on which they form; (*e*) lowered vitality and poor growth of partially affected branches from body

blight or collar blight that fails completely to girdle the affected parts;
(*f*) death of entire tree from body blight or from collar blight that girdles
the trunk near the ground level. The injury may be slight, the crop
may be ruined by blossom blight and the tree survive with little other
damage; or the affected tree may be disfigured, crippled and doomed to
make a struggle for existence; or it may succumb outright.

Fig. 173.—Fire-blight cankers on young active branches.

Etiology.—Fire blight in its various stages is an infectious and con-
tagious bacterial disease due to the specific pathogen, *Bacillus amylovorus*
(Burr.) Trev. Since the early work of Burrill in Illinois in 1878–1881
and Arthur in New York in 1887 establishing the bacterial nature of fire
blight, numerous workers have made successful inoculations with the
bacterial exudate and with pure cultures. The leaf-blight phase of the
disease was not reported until 1915 by the writer, but this has since been
confirmed by various workers, and infections have been shown to occur
also through sepals, petals, stamens, pistil, receptacles and peduncles.

Another infection termed "blast," affecting closed and open flowers, leaves and young fruits and even tender twigs is due to an organism closely related to *Phytomonas syringae*.

The individual bacteria are short rods, averaging 0.6 by 1.5 μ with maximum size of 0.9 by 1.8 μ, while the shortest rods (1 μ) appear almost spherical or oval but are really short and cylindric with rounded ends. The cells are mostly single, often in twos, sometimes in threes or short chains and 2 to 4 flagellate, the flagella being located at or near the poles. No endospores are known. The life-cycle idea of some authors has not been generally accepted. Some differences of the strain prevalent in New Zealand have been recorded by local workers, including a slightly smaller size but more flagella (5 for single rods up to 13 for pairs) and nonpathogenicity to plum, cherry and rose. A recent study of cultures from various portions of the United States and New Zealand, however, has led to the conclusion that the differences do not represent physiological strains. More recently, however, strongly pathogenic strains were shown to be smaller, 0.9 to 1.4 μ long, than the weakly pathogenic strains, 1.5 to 1.7 μ, and these differed in their thermal death point, which was one degree higher for the strongly pathogenic strains (Ark, 1937). Different strains have varied in their ability to ferment or utilize carbohydrates and in the concentration of glucose which they would tolerate.

The blight bacteria are not able to survive the winter in the soil or in the dead parts of their host, since it has been shown that they do not live much longer than several weeks under such conditions. The holdover cankers are the centers from which blight starts in the spring, the bacterial ooze being washed down by rains or carried away by visiting insects. Investigators have found great differences in the percentage of holdover cankers on the different hosts and under different environments. They have even been shown to survive on cankers on severely winter injured limbs. Bacteria from holdover cankers on any host may infect any other suscept, consequently, ornamental plantings and windbreaks may be sources of infection, as well as orchard trees. It has been claimed by some workers that the blight bacteria may overwinter in the beehive and thus be the first source of blossom blight, but recent careful investigations have not substantiated the earlier report, since the blight bacteria were shown to be short-lived in honey or on the body of bees and no blossom blight resulted when a beehive inoculated with the fire-blight bacteria was enclosed with a caged tree.

Since the first proof by Waite in 1898 that bees and flies visiting oozing cankers carry away the bacteria and plant them in the nectar of open blossoms, thus giving rise to blossom blight, it has been shown by other workers that blossom blight may result by direct infection through the

intercellular spaces, regular stomata, nectar-secreting stomata of the nectaries and possibly through hydathodes of the various flower parts. Various other insects including certain species of aphids, several leaf hoppers, the tarnished plant bug, click beetles, ants, bark borers and even such birds as the sapsucker have been recorded as agents in the transmission of the disease. It seems likely that the disease may be spread by almost any sap-sucking or bark-boring insect and, also, through hail injuries and pruning wounds.

The bacteria from any established infection, in blossoms, leaves, twigs or limbs, especially if located well up in the tree, may be washed down by rains and produce new infections. Recent workers seem to attach increased importance to the part played by meteoric water, either direct runoff or wind-blown rain, in the dissemination of blight bacteria from centers of infection and less to the migrations and feeding of insects. It has recenty been pointed out that the nectar of blossoms under arid conditions is too concentrated to permit the multiplication of the bacteria which may be introduced by visiting insects, but the sugar concentration of the nectar is reduced during wet weather, thus facilitating multiplication of the bacteria introduced. On the basis of tests with an artificial nectar, the bacteria grew best with a sugar concentration of 2 to 4 per cent, but decreased with the increased concentration, and ceased to grow at 30 per cent. It has been shown that the fire-blight bacteria may live for two months even in distilled water, for fifty-four days in sterilized soil but a shorter time in unsterilized soil, for nine months in dried exudate, and for two years in infected limbs that have been kept in the laboratory. These facts suggest that holdover cankers may not be the only means by which the bacteria are carried over to the next season. The length of time of retention of viability will vary with exposure to direct sunlight, the temperatures which prevail, moisture relations, the substratum, etc.

Pathological Anatomy.—Diseased tissues show a necrosis or death of cells, the first effects being plasmolysis, digestion of the middle lamellae and later digestion of cell walls, and cell penetration. The bacteria are found in great number in the intercellular spaces of the invaded tissue and cause a separation of cells, sometimes with the formation of pockets filled with bacteria, while in later stages larger cavities may be formed by cell digestion. In stems the cortical parenchyma is invaded first, with deeper tissue later, the bacteria finally reaching phloem, cambium, wood parenchyma and, sometimes, even vessels and pith. The bacteria may accumulate in such quantity that they may be forced to the surface through fissures or ruptures and flow out as the characteristic bacterial exudate; mass mechanical action, enzymes, osmotic pressure and surface tensions play some part in their release.

Predisposing Factors.—*The disease is favored by: (1) a succulent and rapid type of growth; (2) rain followed by warm cloudy weather with a high relative humidity.* The succulent and susceptible host condition may be induced by high atmospheric moisture, abundant rains or heavy irrigation, clean cultivation, and high fertility of the soil, especially from the use of barnyard manure or other nitrogenous fertilizers. According to some experimental tests an increase in the supply of available nitrogen by use of calcium nitrate as a fertilizer will more than double the downward advance of twig blight as contrasted with similar infections on poorly nourished controls. A more resistant condition will result from hot and dry weather, less irrigation, sod or other cover crops and a less fertile soil. Blossom blight will not result unless there are some active cankers producing exudate when the blossoms are open and will vary in amount with amount and type of rainfall and abundance of insect carriers, especially those with sucking mouth parts. The bacteria may be carried from active cankers in an adjacent orchard to another near-by orchard and initiate infections. Pears in the Pacific Northwest generally escape blossom blight, since they flower before holdover cankers are active, but they are frequently active in time to start blossom blight of apples.

Host Relations.—Fire blight is of first importance as a disease of pears and apples, and the quince is also very susceptible. It sometimes occurs on apricots, plums, prunes and cherries. Other hosts under natural conditions are loquat, medlar, wild or cultivated crab, hawthorn, serviceberry, red-berried California holly, American mountain ash, European mountain ash, Japanese flowering quince, fire thorn, wild and cultivated strawberry, raspberry and blackberry, rose and spiraea. The English hawthorn which is used in ornamental plantings or for hedges is very susceptible.

Artificial infections have been successful on many other species of the Rosaceae and on the twigs and nuts of various species of Juglans including the English walnut.

Practically all of our cultivated pears and apples are very susceptible to blight when growing in the nursery but appear to differ somewhat under mature orchard conditions. Bartlett, Howell and Flemish Beauty pears are generally very susceptible while Kiefer, Seckel and Winter Nelis are more resistant. Among apples, Yellow Transparent, Jonathan, Spitzenberg and Wealthy are very susceptible, others moderately susceptible, and Ben Davis, Spy, Gano and Duchess somewhat resistant. Extreme susceptibility is shown by the Transcendent crab and Esopus Spitzenberg. Certain Asiatic species of *Pyrus calleryana* and *P. ussuriensis* brought from China offer some promise as resistant rootstocks to prevent body and collar blight of pears.

Preventive or Control Measures.—The palliative or control measures fall mainly into five categories:

1. *Modification of the Susceptibility of the Host.*—This may be done by use of less irrigation water, cessation of cultivation, the development of a weed cover or a seeded cover crop to utilize the surplus moisture, the avoidance of stimulating fertilizers, the avoidance of excessive pruning, moderate thinning of the fruit, pruning to produce an open or vase type of growth and the removal of water sprouts and fruit spurs near the main trunk or on large branches.

2. *Control or Elimination of Insect Carriers.*—Spraying with Black Leaf 40 for control of aphids has given good results while a 1–3–50 Bordeaux applied when the trees were in full bloom has reduced infection 52 to 79 per cent for apples and 91 per cent for pears. In general the control of sucking, chewing and burrowing insects should give some degree of protection.

3. *The Avoidance of Susceptible Hosts for Windbreaks or for Ornamental Plantings in the Vicinity of Orchards.*—This is emphasized by the New Zealand experience with hawthorn hedges as windbreaks, by the use of the very susceptible English hawthorn as an ornamental, and by frequent cases of holdover canker on neglected quince trees.

4. *Tree Surgery.*—This includes the complete removal of small branches bearing lesions, cutting 4 to 6 inches below the external evidence of the disease, the complete excision of the diseased tissue in case of cankers on large limbs or trunk, and the complete destruction of trees with severe body or collar blight. The pruning tools and cut surfaces should be sterilized with mercury-glycerin disinfectant (see Heald, "Manual of Plant Diseases," 2d ed., p. 357) and the exposed surfaces protected by either Bordeaux paint or asphalt paint. Summer cutting for apple is rarely necessary, but it may be for pears, otherwise the treatment should be made during the period of winter pruning.

A scarification method has been used in California and elsewhere with some success. In this method the dead outer bark is shaved down with a knife or special scraper until most of the diseased tissue is removed over the lesion and for 4 or 5 inches beyond its edges, but not down to the cambium. The cut surface is then painted with the mercury-glycerin disinfectant. This has been reported to be effective on 80 to 95 per cent of the cankers treated.

A more rapid method originated in California has been adopted by growers in other areas. New cankers are painted with a 53 per cent zinc chloride (or weaker 43 and 33 per cent) without any cutting, the strengths being varied according to age of trees, size of branches, and the temperature. This is reported to cure 85 to 90 per cent of treated cankers, but it causes some bark injury. Cadmium sulphate, 15 per cent, is reported to be effective but without injury to the sound tissue.

5. *The selection of resistant varieties* offers some relief, but, unfortunately, many of the most valuable commercial fruits are highly susceptible, especially pears.   The hope for pear growers lies in discarding the susceptible French seedling stocks, and substituting the most valuable resistant stock.   Certain strains of *Pyrus ussuriensis* and *P. calleryana* offer the most promise.

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CROWN GALL AND HAIRY ROOT

Pseudomonas tumefaciens (S. and T.) Duggar
and *P. rhizogenes* (Riker *et al.*)

Crown gall is characterized by the formation of tumorlike enlargements at the crown or on other parts and is known also under various other names such as "crown knot," "root knot," "root tumors," "cane

FIG. 174.—Crown gall on young apple trees.

galls" and "black knot," while hairy root includes the various phases of excessive root production.

Crown gall has been known for more than half a century, being recognized by early French and German writers and by American nurserymen for years before it was the subject of careful scientific investigation. Many different theories were proposed as to the nature of the disease, first as to its nonparasitic origin, but later the evidence pointed to an infectious character. In 1897 Cavara in Italy proved the bacterial nature of the disease on the grape, but this finding was generally overlooked and in 1900 many American workers accepted the report of

Toumey that the causal agent was a slime mold.   A few years later (1907) the work of Smith and Townsend gave the first positive proof in America as to the bacterial origin of the disease and named the organism.   During the earlier years, the hairy-root complex was considered but one of the phases of the crown gall, and it was not until 1930 that Riker and associates demonstrated that typical hairy root is caused by a distinct but

Fig. 175.—Crown gall on roses grown under glass.   Infection through pruning cuts.

closely related species of Pseudomonas.   From the earlier reports up to the present time there has been a constant flow of literature dealing with various phases of these two diseases, and, during the past few years, the flow has apparently increased in volume, amounting to over 100 publications from 1935 to 1941.   These diseases are now known in practically all parts of the world, the crown gall reaching its greatest severity in the warmer regions, while the hairy root is largely limited by the range of the apple, its common host.

   Symptoms.—Two general types of abnormal growth are characteristic of crown gall: (1) typical overgrowths or tumors, *true galls*, of varying form and size, located on the crowns, roots, stems or leaves; (2) excessive or abnormal development of organs either with or without an accom-

panying tumefaction. These galls, almost white at first but becoming darker with age, may be irregular globular or elongated in form with a more or less convoluted surface, the size varying from that of a pea to gigantic overgrowths weighing 50 or more pounds. Most of the tumors are made up of succulent and imperfectly vascularized tissue and have been termed "soft crown galls." These soft crown galls on young woody roots or on herbaceous plants generally decay at the end of the growing season and do not produce roots from their surface. In perennial, woody

Fig. 176.—Blackberry canes showing different degrees of injury from cane galls.

hosts like the apple, the tumors may persist and develop a covering of bark and an interior woody structure, constituting the so-called "hard crown galls." In the light of recent investigations, it seems probable that many of these hard galls are in reality wound overgrowths or callus enlargements. In some hosts the galls are very frequently produced on the aerial parts as well as on the crown or on the roots. This is well illustrated by cane galls or "black knots" on the grape, often 3 to 5 feet above the ground in the form of isolated or elongated excrescences, and by somewhat similar excrescences on the blackberry, especially in the Puget Sound country.

In hairy root there is an abnormal and increased production of roots or of incipient roots, three general types or phases being recognized: (1)

simple hairy root, an abundance of roots with little or no enlargement (a first-season symptom); (2) *hairy or woolly knot,* or somewhat of a tumor-like enlargement with numerous fine roots from its surface or from adjacent parts; and (3) the *aerial form,* appearing on trunk or limbs as smooth fleshy swellings, which develop adventitious roots internally and, with rupture, form warty knots.

In addition to the crown-gall and hairy-root effects, certain non-parasitic abnormalities are recognized which are difficult to determine or to differentiate from those of parasitic origin. The most important of these are burrknots which resemble the aerial form of hairy root; crown-gall enlargements or callus knots from incompatible graft unions; and abnormal root development resembling hairy root, but noninfectious. It has been shown also that abnormal proliferations or neoplasms resembling crown gall may be induced by the action of various chemicals, wire girdles or knife cuts.

Fig. 177.—Hairy-root disease on grafted apple trees. (*After Hedgcock, U. S. Dept. Agr. Bul.* **90**, *Part II.*)

Investigations have shown that aerial tumors may give rise to leafy shoots or flower buds as well as roots, the type of development depending upon the kind of tissues stimulated. The extent to which these fasciations and similar abnormal developments which occur in nature are caused by the two Phytomonas species is uncertain, but experimental results have shown that these bacteria may be possible causal factors in these diseases.

Economic Importance.—The injury from crown gall and hairy root varies with the host, its age at time of infection, location of the tumors, the number of infections and the type of host response. Some of the recorded effects are: (1) retarded growth with dwarfing of root and shoot with development of undersize or chlorotic foliage; (2) the killing of branches, canes or roots from the presence of localized lesions; and (3), in severe cases, the death of the entire plant. The losses involve nursery stock since there is frequently a heavy infection at the time it is dug for storage or delivery; also established plantings in their permanent field location. The present practices call for the rejection of all nursery stock showing any evidences of the disease, and losses up to 70 per cent have been recorded.

It is generally agreed that crown gall is a serious and destructive disease of almonds, peaches, plums, grapes, blackberries and raspberries, but the reports by various workers as to the extent of injury to planted apple orchards are somewhat conflicting, varying from no injury at the end of six years, to interference with food and water transport, mechanical weakening, dwarfing of top and roots, and killing of a certain percentage of the trees. A recent test has shown over one-third of crown-gall apples and nearly one-fifth of hairy-root stock dead at the end of three years.

Etiology.—True crown gall is caused by a specific bacterial pathogen, *Pseudomonas tumefaciens* (S. and T.) Duggar, which was first isolated from the galls on Paris daisy by Smith and Townsend (1904–1906). The pathogenicity of this organism was demonstrated by numerous successful inoculations on various hosts, and it has since been repeatedly isolated from true crown galls on various other hosts by other workers, and typical galls have been produced by inoculations on an extensive list of hosts, including herbaceous plants and fruit, nut and shade trees. Studies during the last few years have shown that many of the overgrowths on apple-nursery trees are not really parasitic crown gall but graft misfits.

In callus or graft knots, the periderm is normal and the tannin test is negative, while in crown gall there is a surface zone of dark, polyhedral, closely packed cells and the tannin test is positive.

Several different workers showed that there were marked differences between the apple organisms causing typical crown gall and the ones responsible for woolly knot and hairy root, and this differentiation of the two forms (Riker, *et al.*, 1930) culminated in the recognition of the hairy-root organism as a new species, *Pseudomonas rhizogenes*. This organism was isolated from 78 of 96 enlargements of the hairy-root and woolly-knot type, and it reproduced typical hairy root by inoculation, followed by the recovery of the organism. It has also been shown by inoculations that the same organism will cause the aerial form of hairy root (burrknot), but nonparasitic forms of both hairy root and burrknot are recognized. The crown-gall hairy-root problem is complicated by mixed infections involving one or both of the pathogens with the noninfectious overgrowths.

Both of the organisms are small rods of slightly different size, producing small, slow-growing, circular, raised, glistening, translucent colonies, and can be differentiated by a number of cultural tests. Both are motile by polar flagella and this character appears to vary under different conditions according to the observations of various investigators.

The crown-gall and the hairy-root bacteria are intercellular, and occur in large numbers especially in the surface and in subsurface parenchyma,

and are able to advance in the intercellular spaces as zoogloeal strands. It has been contended that this advance and also advance in vascular tissue may account for secondary tumors distant from the first point of infection.

The presence of the bacteria on the surface of developing galls will explain a number of features: (1) the difficulty of isolating the causal organism from the interior of galls; (2) the continued meristematic activity of the tissues close to the surface; (3) the ease with which soil may become contaminated since water must wash many of the surface organisms into the soil. The bacteria may reach wounds directly from contaminated soil in many ways: by irrigation waters or splashing and washing from rains, by contaminated pruning tools, by insect carriers or possibly by wind-borne soil. The entrance through grafting wounds will explain the frequent position of large tumors at the crown of the apple and other fruit trees.

The presence of the bacteria does not kill the affected tissue but stimulates the cells and thus causes an abnormal and rapid cell division, some cells being reduced in size, and others enlarged, leading to the formation of tumors or to the abnormal development of organs. The period of incubation before the appearance of an evident gall varies from five days to several weeks on various hosts, and, in some cases, the organism may remain dormant for months before any evidence of an infection can be noted. The gall formation is dependent upon the growth of the host. In addition to the formation of the typical galls certain virulent strains have been reported to induce the following: (1) increased epinasty of petioles; (2) increased production of adventitious roots; (3) increased cambial activity; (4) retarded development of certain buds; and (5) delayed abscission of senescent leaves.

The crown-gall and hairy-root bacteria are very widely distributed organisms and are apparently native in many soils, where they may lead an independent life or persist in old host lesions. They may overwinter in the field or in the storage cellar and may be transmitted with seedling roots. Both species appear to be unable to enter normal uninjured tissue, but they can readily establish themselves through mechanical injuries of many types. The development of infections is influenced by the temperature, the soil moisture and the soil composition or reaction. *Pseudomonas tumefaciens* is intolerant of acid conditions of the soil.

The claim of some investigators that the crown-gall organism is a polymorphic form passing through a life cycle including a filterable-virus stage has not been substantiated by recent studies. The literature is replete with comparisons of crown gall (plant cancers) to malignant tumors in man, but true homologies have not been substantiated. .

Secondary tumors have been noted to develop at varying distances from the primary infection in certain hosts. These are believed to result from bacteria carried upward in the vascular system. Such secondary tumors may be internal in association with broken vessels or become external as a result of wounds which break the vessels and liberate the bacteria, or extruding or surface tumors may be formed when a rootlet forces its way to the surface and becomes disorganized into a tumor.

Galls have been formed by inoculation with indolacetic acid and by the ether extract of crown-gall cultures, but it is doubtful if this or any similar chemical entity plays a major part in the initiation of crown gall. It is interesting to note that crown-gall tumor tissues have been grown in vitro free from crown-gall bacteria and have continued to grow and maintained their virulence through 10 successive transfers.

Host Relations.—Crown gall has been found to occur naturally on the pome fruits, all the stone fruits, various species of Rubus (blackberries, raspberries and loganberries), currants and gooseberries, grape varieties, nut trees, such as walnuts, pecans and almonds, numerous woody and herbaceous ornamentals, several deciduous-leaved trees, alfalfa, cotton, beet, castor bean, turnip, salsify, parsnip and hop. In addition to the natural infection, many herbaceous and woody hosts have been successfully inoculated with pure cultures, and a wide range of susceptibility has been shown among the different species and in some cases in different varieties of the same species.

Hairy root is primarily a disease of apples, but the causal organism has been isolated from several species of spiraea and positive inoculations have been secured on a number of annual plants, herbaceous perennials, shrubs and trees, which would indicate a probable wider natural range than has been recorded.

The following statement applies to crown gall: Myrobalan, peach, apricot and almond stock are very susceptible (94 to 97 per cent) as contrasted to plums and prunes (10 per cent), but promising resistant rootstocks of both Prunus and Amygdalus have been found; the cherry is more resistant than other stone fruits; the California black walnut is more resistant than English walnut; American grapes are more resistant than European varieties; and variations have been noted for apple varieties, but comparisons have shown different resistance of the same varieties in different regions. It has been suggested that the basis for resistance to the crown-gall organism is the acidity of the cell sap, since many immune plants have a cell sap acidity of more than pH 5.70.

Prevention and Control.—The following practices are of value and should be given consideration in the general control program:

1. *The Use of Disease-free Stock.*—Nursery stock showing evidences of either type of bacterial infection or the noninfectious types of overgrowths should be discarded. Do not replant small fruits or other suscepts from diseased plantations. Some advise use of stock if galls are on lateral roots which can be pruned off without too great mutilation. Remove orchard trees the first year if crown infections develop.

2. *Sanitary Practices.*—The following have been emphasized: (*a*) sterilization of seedbeds; (*b*) sterilization of soil for transplants; (*c*) treatment of nursery stock before delivery by immersion of the root and crown in a fungicide (copper sulphate or a sand-argillaceous earth-Uspulun bath); (*d*) avoid mixing diseased and healthy stock at digging time; (*e*) in replacing infected stock, either sterilize the soil or use uncontaminated earth for filling; (*f*) destroy all material showing evidences of infection; (*g*) avoid crown wounds in the nursery and in the orchard. Some workers have reported that an interval between root pruning and planting reduces infection and also recommend the disinfection of the soil at planting time with an organic mercury (uspulun etc.) and advise the dipping of the pruned stocks in 1 per cent uspulun or substitute prior to planting. It is claimed that the efficiency of this treatment is increased manyfold in acid soil. It is worthy of note that according to tests by Siegler (1940) pits dipped in an aqueous suspension of calomel (4 ounces per gallon) gave only 6 per cent of galls as contrasted to 71 per cent in the untreated controls. Elgetol (sodium-dinitro-cresol) diluted with methyl alcohol (20–80) painted on clean galls and $\frac{1}{2}$ to 1 inch of healthy bark has killed the galls (Ark, 1941).

3. *Surgery.*—Removal of tumors on established trees followed by disinfection of cut surfaces has been advised by some workers, but this practice is of doubtful value.

4. *Rotation or the Selection of Clean Ground.*—Important for stone fruits and the more susceptible hosts; less important for apples. Some recent studies have shown that the pathogen can live for at least two years in the soil without the presence of any susceptible host. The astounding claim has been made by another investigator that the bacteria have persisted in grainland for 40 years when no susceptible hosts had been grown (Cochran, 1941).

5. *Care in Making Grafts and Their Protection or Sterilization.*—The exact fitting of stock and scion is of prime importance, for lessening not only the noninfectious overgrowth, but also the parasitic infections. Various details of operation designed to reduce infections have been emphasized including: (*a*) the use of clean, washed and dry understocks for grafting; (*b*) dipping in a fungicide before grafting or after (mercuric chloride 1–1000, one week before); Bordeaux 8–8–50; Semesan, 1 to 400; or puddling in the fungicide, soil and water; (*c*) the use of insect repellents

to keep the grafts free from root-chewing insects; (d) budding instead of grafting (Gloyer, 1934); and (e) care in fitting and wrapping the grafts. Various wraps have been recommended including cloth, string, adhesive tape and nurserymen's tape treated with 1–1000 mercuric chloride; the latter having given good commercial control for apple stock.

6. *Use of Resistant Varieties or Resistant Stocks.*—Selection when consistent with the horticultural needs is especially applicable to the stone fruits.

It is interesting to note that some recent work has shown the possibility of killing tumors by injection (osmic acid), and by radiant heat. Prevention and cure have also been claimed by vaccinotherapy, using injections or applications of emulsions of young, heated agar cultures.

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IMPORTANT DISEASES DUE TO BACTERIA

For key references to these diseases see F. D. Heald, "Manual of Plant Diseases," 2d ed., pp. 385–388.

Principal host	Common name of disease	Scientific name of causal organism
Apple, pear, etc....................	Fire blight	*Bacillus amylovorus* (Burr.) Trev.
Plum, peach......................	Black spot	*Pseudomonas pruni* E. F. S.
Pome fruits, stone fruits and others....	Crown gall	*P. tumefaciens* (S. and T.) Duggar
Pome fruits, stone fruits and others....	Hairy root	*P. rhizogenes* Riker *et al.*
Grape...........................	Black knot	*P. tumefaciens* (S. and T.) Duggar
Stone fruits......................	Bacterial gummosis	*P. cerasus* Griffin
Apple...........................	Blister spot	*P. papulans* Rose
Banana..........................	Blood disease	*P. celebense* Gäumann
Citrus fruits.....................	Citrus canker	*P. citri* Hasse
Citrus fruits.....................	Blast and black pit	*P. syringae* Van Hall
Olive............................	Tubercle	*P. savastanoi* E. F. S.
Bean............................	Blight	*P. phaseoli* E. F. S.
Crucifers........................	Black rot	*P. campestris* (Pam.) E. F. S.
Carrot and other vegetables.........	Soft rot	*Bacillus carotovorus* L. R. Jones
Cauliflower......................	Spot disease	*Pseudomonas maculicolum* (McC.) Stev.
Cucumber.......................	Angular leaf spot	*P. lachrymans* (S. and B.) Carsner
Cucurbits........................	Wilt	*Bacillus tracheiphilus* E. F. S.
Lettuce..........................	Leaf diseases	*Pseudomonas marginalis* (Brown) Stev.
		P. viridiliridum (Brown) Stev.
		P. vitians (Brown) Stev.
Lettuce..........................	Rosette	*Bacterium rhizoctonia* (Thomas) Stapp
Peas............................	Bacterial blight	*Pseudomonas pisi* Sackett
Solanaceae.......................	Brown rot	*P. solanacearum* E. F. S.
Potato...........................	Blackleg	*Bacillus atrosepticus* Van Hall
Potato...........................	Ring rot	*Bacterium sepedonicum* S. and K.
Tomato..........................	Bacterial canker	*Bacterium michiganense* E. F. S.
Tomato..........................	Bacterial spot	*Pseudomonas vesicatoria* (D.) Stev.
Sweet corn.......................	Bacterial wilt	*Bacterium stewarti* (E. F. S.) Stev.
Cotton..........................	Angular leaf spot	*Pseudomonas malvacearum* E. F. S.
Sugar cane.......................	Bacterial gummosis	*P. vascularum* (Cobb) E. F. S.
Sugar cane.......................	Java gum disease	*P. albilineans* (Ashby)
Sugar cane.......................	Red-stripe disease	*P. rubrilineans* (L. P. B. M.)
Sugar beet.......................	Bacterial pocket	*P. beticola* (S. B. and F.) Stev.
Tobacco	Wild fire	*P. tabaci* (W. and F.) Stev.
Tobacco..........................	Black or angular leaf spot	*P. angulata* (F. and M.) Stev.
Barley...........................	Bacterial blight	*Pseudomonas translucens* (J. J. and R.) Stev.
Corn............................	Bacterial stalk rot	*P. dissolvens* Rosen
Corn, sorghum, etc.................	Bacterial spot	*P. holci* Kendrick
Oats, barley, rye and wheat.........	Halo blight or blade blight	*P. coronafaciens* (Elliott) Stev.
Oats.............................	Stripe blight	*P. striafaciens* (Elliott).
Wheat...........................	Black chaff	*P. translucens* var. *undulosum* (S. J. and R.) Stev.
Wheat...........................	Basal glume rot	*P. atrofaciens* (McC.) Stev.
Alfalfa..........................	Bacterial stem blight	*P. medicaginis* Sackett
Alfalfa..........................	Bacterial wilt and root rot	*Bacterium insidiosum* (McC.) Stapp
Clover..........................	Bacterial leaf spot	*Pseudomonas trifoliorum* (J. W. W. and McC.) Stapp
Geranium........................	Leaf spot	*Pseudomonas erodii* Lewis
Gladiolus........................	Scab	*P. marginata* (McCulloch)
Hyacinth........................	Yellow disease	*P. hyacinthi* (Wakker) E. F. S.
Larkspur........................	Black spot	*P. delphinii* (E. F. S.) Stapp
Lilac............................	Blight	*P. syringae* Van Hall
Sweet pea.......................	Streak	*Bacillus lathyri* Manns and Taub.
Coconut, etc.....................	Bud rot and soft rot	*B. coli* (Esch.) Mig.
Mulberry........................	Blight	*Pseudomonas mori* (B. and L.) Stev.
Aleppo pine......................	Tuberculosis	*P. pini* (Vuill.) Petri
Walnut..........................	Blight	*P. juglandis* Pierce

CHAPTER XIV

PARASITIC SEED PLANTS AND THE TROUBLES THEY CAUSE

PARASITIC SEED PLANTS

Many flowering or seed plants are parasites or half parasites and cause more or less disturbance in the life of their hosts. When crop plants are concerned, they may cause serious injury. Some have a chlorophyll apparatus while others have none, but all are robbers, stealing all or a part of their food from their hosts.

Groups of Parasitic Seed Plants.—Of the total number of seed plants, only a few have degenerated to the robber class, and of these only a few are of importance because of their relations to plants of economic importance. The following groups may be recognized on the basis of physiological behavior:

1. Herbaceous plants bearing green foliage and rooted in the soil but provided with haustorial disks, which are attached to the roots or crowns of their hosts. Examples: Sandalwood of India (*Santalum album*), bastard toadflax (*Thesium alpinum*), Commandra species, eyebright (*Euphrasia spp.*), the yellow rattle (*Rhinanthus spp.*), cow wheat (*Melampyrum arvense*) and louseworts (*Pedicularis spp.*).

2. Underground plants bearing leaves without chlorophyll, and with haustorial disks attached to the roots of trees and shrubs. Example: the toothwort (*Lathraea squamaria*).

3. Foliage-bearing, chlorophyll-containing, bushy, perennial plants growing upon the aerial portions of various trees. Examples: the giant mistletoes (*Loranthus spp.*), European mistletoe (*Viscum album*), the American mistletoes (*Phoradendron spp.*), the dwarf or scaly mistletoes (*Razoumofskya spp.*) and some Santalaceae of the East Indies.

4. Plants with no chlorophyll and only a few rudimentary scale leaves on filiform twining stems without soil connection, but provided with sucking organs or haustoria which absorb both crude and elaborated foods from the host. Examples: the woevine and other species of Cassytha and the dodders (*Cuscuta spp.*).

5. Plants without chlorophyll and with their tissues merged with those of their host to form a more or less tuberous enlargement from which the aerial, flowering stems arise. These may be almost naked or provided with scattered rudimentary leaves or closely imbricated scalelike

leaves. Examples: the broom rapes (Orobanchaceae) and the Balano-phoraceae, on the roots of trees in equatorial regions.

6. Plant body merged with the tissues of the host between the wood and cortex to form a more or less hollow cylinder, from which the flowers are formed and finally burst through the cortex. Examples: Various species of the tropical and subtropical family, the Rafflesiaceae. The genus Rafflesia is noteworthy as including *R. arnoldii*, a native of Sumatra parasitic on the roots of vines, and claimed by some writers to produce the largest flowers in the world (1 meter diameter).

DODDER OR LOVE VINE

Cuscuta spp.

The dodders or love vines are of importance as pests of clover, alfalfa and flax but are of minor concern as parasites of various other cultivated plants. Some of the common names are "strangleweed," "goldthread," "hairweed," "pulldown," "hailweed," "devil's-hair," "devil's-ringlet," "devil's-guts" and "hell-bind."

Dodders belong to the single genus, Cuscuta, of the Cuscutaceae, a family very closely related to the Convolvalaceae or morning glory family. The ancestral habit of twining around other plants for support led to the habit of parasitism. The most recent monograph by Yuncker recognizes 158 species on a great variety of hosts.

Important Species on Crop Plants.—The following species attack clover, alfalfa and some other legumes although not confined to these hosts:

1. Clover dodder, *Cuscuta epithymum* Murr.
2. Small-seeded alfalfa dodder, *Cuscuta planiflora* Ten.
3. Large-seeded alfalfa dodder, *Cuscuta indecora* Choisy
4. Chilean dodder, *Cuscuta suaveolens* Seringe
5. Peruvian dodder, *Cuscuta odorata* R. and P.
6. Field dodder, *Cuscuta arvensis* Bey.
7. Common dodder, *Cuscuta gronovii* Willd.

Numbers 1, 2, 4 and 5 are emigrants from foreign countries, while Nos. 3, 6, and 7 are natives of North America. Numbers 6 and 7 infest many species of native hosts showing but little preference for any one. Number 5 has been reported from Europe but not from America.

Cuscuta epilinum Weihe, flax dodder, attacks flax, hemp and some other hosts but never clover or alfalfa.

Cuscuta europea L. is common on hops, vetches, sugar beet, potato, etc., in Europe but is relatively rare in America.

The Parasite.—The various species of dodder are so similar that a single description of their general characters will suffice. When dodder

first becomes evident in the field, it will be noted as a tangle of branched threadlike leafless stems, devoid of chlorophyll, free from the soil and twining around the stems or other parts of its host or forming an inter- lacing mat. The common color is yellowish or orange, but stems of certain species may be almost white or even tinged with red or purple.

FIG. 178.—Dodder on alfalfa.

The leaves are represented by minute functionless scales, and the roots by sucking organs or *haustoria* which are formed on the concave side of the coils of twining stems, following the stimulus of contact with the host. The tiny white, pink or yellowish flowers occur in clusters on the twin- ing stems from early June until the end of the growing season. The fruit capsules mature seed from July until frost, and as many as 3000 seeds have been counted from a single plant.

Under favorable conditions of moisture and temperature, the seeds of dodder germinate to form a slender, yellowish, unbranched thread, generally larger at the lower end, which may remain for a time either in contact with the soil or slightly embedded in it. The distal end is raised

Fig. 179.—Diagrammatic representation of the complete life cycle of dodder from the seed (*a*) to the maturing of a new crop of seed. (*After Hansen, U. S. Dept. Agr., Farmers' Bul.* **1161.**)

more or less and as it grows in length, moves in a circle in its search for a support. If a susceptible host stem is reached, the young dodder stem twines around it and soon forms haustoria which penetrate the host tissue. The young stem, below the first stem coil with the haustoria,

shrivels and dries up, and from now on the dodder lives entirely on the food absorbed from its host, as it branches and spreads in an ever-increasing tangle over its host stems.

A recent study (Dean, 1937) has called attention to the production of galls on the host by at least five different species of Cuscuta. Those produced at the point of initial attack are designated as *primary* and are typically short bulbous enlargements, while those at other points are classed as *secondary* galls. These may resemble the primary galls or they may be elongated or irregular. Some of the galls may increase in size sufficiently to break the dodder stem. Dodder has also been noted to

Fig. 180.—Haustorium of *Cuscuta epilinum*   *E*, epidermis; *R*, cortex; *H*, wood of flax stem; *e, r* and *g* are tissues of Cuscuta. Semidiagrammatic. (*After Sachs.*)

cause hypertrophy of fruits (peas and beans) as well as stems (Dean, 1939).

Without giving details of the development and penetration of the haustoria into the host stem, it will suffice to say that xylem and phloem elements of the haustoria finally come into physiological continuity with xylem and phloem elements of the host stem, thus affording a pathway along which both crude and elaborated food may be transported from the host stem into the stem of the parasite.

Effect of Dodder.—In clover or alfalfa the dodder is introduced with the seed and first appears in the field in circular spots, the size depending on the age of the infestation, which will spread radially from year to year. The maximum injury in most cases results during the second or third year after seeding, but the damage will vary with the species of dodder, the amount of seed introduced, and the host conditions. In

long-established infections the dodder is not much in evidence in the early spring, but later develops the characteristic tangled masses of twining stems around the margin of the spots. Cases are on record in which fields have been completely ruined by dodder, but even with less severe infestation the quantity and quality of the hay crop is lowered, while it is of most concern in fields grown for seed production.

Fig. 181.—Seed of dodder compared with alfalfa seed *A*, clover dodder (*Cuscuta epithymum*); *B*, small-seeded alfalfa dodder (*C. planiflora*); *C*, field dodder (*C. arvensis*); *D*, large-seeded alfalfa dodder (*C. indecora*). Dodder seeds are small and rounded as compared with alfalfa seeds, which are large and irregular. (*After Hillman, U. S. Dept. Agr., Farmers' Bul.* **306**.)

Etiological Relations.—Susceptibility of a host to dodder is influenced by the hydrogen-ion concentration of its cell sap, its sugar content and the formation of oxidation ferments. Too great a divergence of these factors in host and parasite is unfavorable to a parasitic relation.

Dodder overwinters in two ways: (1) by seed which remains dormant until spring; and (2) by established stems on perennials. It has been shown that the clover dodder (*Cuscuta epithymum*) on alfalfa is perennial, and that overwintered infestations are especially important since the species is a poor seed-producer in the United States. Even minute portions of dodder stems, if still retaining haustorial contact with the host, can continue the infection, and even separated fragments of dodder under moist conditions may establish new connections.

Dodder may be introduced or disseminated after introduction into a field: (1) with the seed; (2) by irrigation water; (3) by hay from infested fields; (4) by contaminated manure; or (5) by farm operations and the movement of livestock. Dodder seed in commercial seed of alfalfa or clover may originate from two sources: (a) from seed threshed from infested fields; and (b) mixing of screenings of low-grade seed with a clean crop, a practice of unscrupulous dealers. The contaminated seed is generally the first source of infections but after that the other means of spread are operative.

Control and Eradication.—First attention should be given to practices to prevent introduction of dodder, and if present to guard against its spread by: (1) the selection of dodder-free seed; (2) the avoidance of dodder-infested hay; (3) preventing the movement of grazing animals from infested to clean fields; (4) restriction of the flow of irrigation water so as to avoid passing through infested areas; and (5) the avoidance of the use of dodder-containing manure.

The small-seeded dodder may be separated from clover or alfalfa on the farm by hand sieves, with good results by the use of a (20 meshes to the inch) brass screen, using No. 30 to 34 gauge wire, but this is not effective for the large-seeded dodder. The separation of dodder from commercial seed is now generally done by seed companies provided with special cleaning machinery. Three types of cleaners are in use: (1) power-driven graders with special screens; (2) the Dosser machine, in which the velvet linings retain the smaller seed that cannot be screened out; and (3) by an electromagnetic process, in which the dodder seed, coated with a powder, is held by a magnet, and thus separated from the legume.

Eradication of established infestations should be initiated before maturing seed if possible. In this case patches should be mowed early before the flowers have opened, and small patches may be further protected by sprinkling the cut hay with crude oil or kerosene and burning. After seeding more care is necessary. Some of the methods used are: cover infested area with straw and burn; sprinkle with kerosene or crude oil and after a few days ignite; burn infested areas with a blowtorch; and burn after cutting and drying. In severe infestations the ground should be fallowed after the selected eradication measure has been completed, and then followed with a five-year rotation beginning with a nonleguminous tilled crop.

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THE AMERICAN MISTLETOES

Phoradendron spp.

These leafy, green parasites of forest and shade trees are known for two reasons: (1) because of their use in home decoration during the Christmas season, and (2) because of the serious injury which certain species cause to forest, nut and shade trees.

The genus Phoradendron is strictly American, the various species ranging from Oregon, southern Colorado, the mouth of the Ohio River and southern New Jersey southward into Mexico, Central America, the West Indies and in South America to the mouth of the La Plata. A late monographic revision by Trelease recognizes 240 species. Most of the hosts are angiosperms but several species parasitize coniferous hosts. Mistletoe has attracted most attention as a pest in Texas and other portions of the Southwest, in the transition zone between the humid Gulf states and the arid Southwest. The common European mistletoe is *Viscum album*, a species very similar in general appearance to the American mistletoes.

The Parasite.—Most of the species of Phoradendron show a marked similarity in growth and general habit, appearing as bunched tufts of leafy, perennial, suffruticose shoots on the branches of their hosts. Because of their evergreen character, they present a striking picture when their hosts are devoid of foliage. A few species form long, pendant tufts or wide-spreading, fountainlike masses.

The shoots are well supplied with opposite, expanded leaves, which are petioled or petiolately contracted, but, in a few species, they are reduced to scales. There is a well-developed chlorophyll apparatus in both leaves and stems, by which the parasite is able to manufacture its own carbohydrate food, but the plants, especially during the winter, have a slightly yellowish-green cast, in some species even a golden coloring (*Phoradendron macrophyllum*) or an olive or brownish shade (*P. juniperinum*, etc.). The stem of the mistletoe expands in the cortex of the host into an irregular branched structure, the *haustorium*, from the underside of which peglike outgrowths, the sinkers, are formed which penetrate to the cambium and later, by the formation of the annual rings of xylem, come to be embedded in the wood.

The haustorium and sinkers fix the parasite in position and serve as an absorbing organ. The sinkers come into direct contact with only xylem cells of the host, hence the parasite cannot rob its host of elaborated foods, which must travel along the phloem. The mistletoe is then a

FIG. 182.—A typical growth of *Phoradendron engelmanni* about 2 feet in height. (*After York.*)

FIG. 183.—Mistletoe plants five, six and seven years of age. (*After York.*)

water parasite, that is, it obtains its water and mineral salts from its host but elaborates its own food in the same way as an ordinary green plant. The haustorium gives rise to buds on its upper surface, which may produce new shoots at some distance from the primary shoot. If the aerial shoots are broken off, the cortical haustorium will immediately give rise to new shoots from dormant buds.

The northern mistletoes are dioecious, with axillary spikes of three- or sometimes two-, four- or five-merous, inconspicuous, apetalous flowers.

Fig. 184.—Cross section of a branch infested with mistletoe. *m*, shoot of mistletoe; *h*, haustorium; *s*, sinker; *c*, cortex of host.

The ovary is inferior, one-celled, one-ovuled and develops into a more or less globose berry with a single albuminous seed and a very viscid mesocarp. The mature berries of the common species are whitish translucent, or sometimes shaded with greenish yellow, while the legume mistletoe (*Phoradendron californicum*) and its conifer-inhabiting allies have straw-colored or reddish berries.

Effect upon the Host.—Based on the studies of mistletoe in the Southwest, some of the reported effects are: (1) more or less hypertrophy of the branch at the point of infection; (2) atrophy and final death of a branch beyond a mistletoe tuft; (3) deforming of branches or even of the main trunk owing to infections of long standing; (4) the excessive formation of shoots by the host, giving rise to a sort of witches'-broom effect, as in many infections on the osage orange; (5) delay of the host in putting out its leaves in the spring; (6) retardation of growth with its stunting and dwarfing effect. Doubt has been expressed by some writers as to whether mistletoe ever kills its host, while others have shown that it not

only·causes mechanical injury, but saps the vitality and in heavy infestations of certain hosts may result in death of the entire tree.

Etiological Relations.—The fleshy, viscid berries and the seeds they contain are disseminated very largely by birds, for the most part "by being wiped from the beak against a branch in the bird's efforts to remove the adhesive pulp" or also through the excrement. "In either case the pulp still remaining about the seed causes it to stick to the branch and by drying to become firmly pasted there." Mockingbirds, cedarbirds or waxwings, cardinals, robins and sparrows are reported to be important agents of dissemination, the first being named the chief distributor. Later in the spring when the mistletoe berries soften, they may be broken from their attachment by winds or rains, and fall to other branches on which they become crushed.

By February or later the seeds germinate, the exact time, of course, depending upon the temperature and moisture. The radicle turns toward the substratum and, when it comes in contact with it, enlarges and becomes flattened to form a more or less circular *attachment disk*. This develops a *primary haustorium* from its under surface and, after penetrating into the bark, spreads out in the cork cambium. Sinkers are formed which reach the wood, while buds of the first shoots are formed on the attachment disk. The rate of growth is very slow, and, by the end of the second year, the shoot has produced its first pair of foliage leaves. Mature plants of the Texas mistletoe (*Phoradendron engelmanni*) may reach a length of 1 to 3 feet and, in specimens of maximum size, have been estimated to be at least twenty years old. With continued growth, the cortical haustorium may completely encircle a branch, and new shoots may appear even on the side opposite the original point of entrance. There appears to be no fixed limit to the continued existence of mistletoe on its host.

Physiological Strains.—The European mistletoe (*Viscum album*), shows three different strains or races: (1) the pine mistletoe on pines and some other hosts; (2) the fir mistletoe on Abies species; and (3) the mistletoe of broad-leaved trees. The American mistletoes have not been studied so intensively and thoroughly as *V. album*, but it is stated that *Phoradendron flavescens*, the eastern mistletoe, most generally affects only one of its numerous hosts in a given region. Texas mistletoe has been recorded on 32 different hosts, and it seems likely "that the central Texas form of mistletoe may be more or less freely established upon all of the hosts by seed carried from the mistletoe growing upon any one of them." The marked similarity of several of the recognized species, whose separation has been based largely on the study of herbarium specimens, suggests that more detailed field studies and cultures might be undertaken with profit.

Control.—Principal attention is directed to the control of mistletoe on ornamental shade trees many of which in certains locations in the southern United States are heavily infested. It is of minor importance on fruit and nut trees. If trees are carefully watched, young infections of mistletoe can be removed by ordinary pruning operations, cutting the branches off a few inches below the point of infection. In established infestations which have been permitted to develop unmolested two courses are open:

1. The breaking of the mistletoe, which retards its growth and gives some relief to the tree. This can be accomplished in tall shade trees by means of a mistletoe hook, consisting of a curved or L-shaped iron inserted in the end of a long pole, which is used to pull or break the mistletoe tufts from their point of attachment. The haustoria will produce new shoots with the resumption of growth, but a repetition of the breaking will reduce the damage.

2. The cutting out of the mistletoe with treatment of the haustoria to prevent the formation of new shoots. Complete killing can only be assured when the bark covering the haustorium is cut away together with the external parts of the haustorium and the cut surface treated with creosote or coal tar or a combination of the two. Even this treatment may not be successful in old infections with widely ramifying haustoria.

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THE SCALY OR DWARF MISTLETOES

Razoumofskya spp.

The scaly or dwarf mistletoes parasitize the branches and upper trunk of various conifers. The plants bear scalelike leaves containing chlorophyll but are most frequently of a pale or yellowish-green color. They vary in size from tiny plants only $\frac{1}{2}$ inch tall to bushy plants 6 inches or slightly more in length. Some of the effects are killing of young trees, retarded growth and the formation of burls and witches'-brooms. While a few species occur in Europe and Asia, they appear to be of most importance in the coniferous forests of the Pacific Northwest.

The following are the principal American species of importance on conifers:

Razoumofskya americana (Nutt.) Kuntze on lodgepole pine.

·*R. campylopoda* (Eng.) Piper on western yellow pine and other two- and three-needled pine.

R. cyanocarpa (A. Nel.) Ryd. and *R. cryptopoda* (Eng.) Cov.

R. douglasii (Eng.) Kuntze on the Douglas fir.

R. douglasii abietina (Eng.) Piper and *R. occidentalis abietina* (Eng.) Coville on the true firs.

R. laricis Piper on western larch.

R. pusilla (Peck) Kuntze on spruce and larch in northeastern states.

R. tsugensis Rosend. on hemlock.

FIG. 185.—Scaly mistletoe (*Razoumofskya sp.*) on pine.

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CHAPTER XV

DISEASES DUE TO NEMATODES

NEMATODES

Nematodes live free either in moist earth or in water, in decaying vegetable or other organic substances or as ecto- or endoparasites on plants or animals. They feed mostly on juices which they suck up directly from the organic materials of the substratum or which they make available from living cells by boring into them with a buccal spear.

General Characters of Nematodes.—The nematodes, or nemas, sometimes called roundworms, have tubular or filiform bodies, with mouth and well-developed alimentary canal. The mouth is provided with papillae or lips or with hooks or spines in the oral or buccal cavity, and leads into a narrow oesophagus, which usually has thick muscular walls and a cuticularized lining and may be dilated into one or more muscular oesophageal bulbs or pharynx. By valves and a muscular wall, the oesophagus pumps in fluid food as well as, in some species, solid particles and, by peristaltic action, passes it on to the intestine, usually a straight tube which opens near the posterior end of the body on the ventral surface.

The body wall is muscular, and encloses a body cavity containing the blood fluid, the alimentary canal, and the excretory and reproductive organs. There is no definite circulatory system, and respiration organs are lacking. The body is unsegmented, but the stiff cuticle is often transversely ringed. The muscular body wall makes it possible for the body to be knotted, curved or bent, and permits the characteristic undulatory movements.

The sexes are generally separate and the males smaller than the females. The females lay eggs but in a few cases they may bear living young. Many species are parasitic during either all or a part of their life cycle, and some animal parasites require two entirely unrelated hosts to complete their life cycle. Notable parasites of man and animals are: *Trichinella spiralis* (Trichina) the cause of trichinosis in man, a disease which is contracted by eating uncooked pork in which the worms are encysted; *Filaria medinensis* (Guinea worm) which, in the tropics of the Old World, forms subcutaneous abscesses in man; and *Necator americanus* (Hookworm), the cause of the hookworm disease, a terrible scourge among the Negroes and poor whites of the southern United States.

Classification of Important Plant Nemas.—The plant pathologist is interested in but one family of the nematodes, the *Anguillulinidae* (*Tylen-*

chidae). This is a family of minute forms which live either free in the soil, decaying organic matter, water, etc., or on or in plants as parasites. The following genera are the most important ones furnishing plant parasites: *Anguillulina, Tylenchulus, Heterodera* and *Aphelenchoides*.

ROOT KNOT OR ROOT GALL

Heterodera marioni (Cornu) Goodey

This disease is known by several other common names such as "root gall" and "big root" because of its effects and as "eelworm disease" because of the causal organism.

The root-knot nematode, a native of the tropical and semitropical regions of the Old World, has become world-wide in its distribution. Its range in the open is limited in large part by its temperature requirements, which confine it mainly to the warmer regions, but it frequently is very destructive to certain glasshouse crops at any latitude. It is common and often serious on field-grown crops in the South Atlantic and Gulf states and California, but occurs sporadically as far north as the Canadian boundary.

Symptoms and Effects.—The manifestations of the disease and its effects are: (1) the galls or enlargements on the root system; (2) dwarfing and retarded growth with more or less root killing and reduction of yield; (3) a paler green color of the foliage than normal, frequently accompanied by marginal necrosis; (4) a more pronounced wilting in hot, dry weather than for healthy plants; (5) the destruction of seedlings almost as soon as they get above the surface of the soil, very similar to fungous damping-off, especially in heavily infested soils; and (6) the premature death of older plants or even of those approaching seasonal maturity. The degree of expression of these effects is generally an indicator of the size and abundance of the root galls. Very marked differences in effects are exhibited by the various hosts, some for example, the mulberry, are not damaged seriously by numerous galls, while others as illustrated by okra are quickly killed by a comparatively few galls.

The root galls or enlargements may appear as slight swellings, as small, scattered, tuberclelike growths or as extensive swellings 1 or 2 inches in diameter on either large or small roots and, in extreme cases, may involve nearly the entire root system (see Fig. 186). The galls on violets are the smallest, and the largest have been recorded on roses (size of duck eggs). The galls on dicots are, most frequently, sharply defined tubercles, while on monocots they are generally slender spindle-formed enlargements. If the knots are cut across, from one to several dark specks may be noted in the cortex, marking the location of the pathogen. In the Irish potato, the tuber suffers more than the roots

and when heavily infested may show a rough, warty surface, but in light infestations the presence of the nematodes may not be evident until the tubers are cut. Somewhat similar characters are to be noted in fleshy roots such as carrots or turnips. Such tubers or roots when cut across will show small, brown, necrotic spots just below the skin (Fig. 187), in which either young larvae or enlarged gravid females may be found. Affected organs, if carefully dissected or broken open, may show the

Fig. 186.—Roots of tomato plant showing severe invasion by root-knot nematodes. (*After G. F. Atkinson.*)

enlarged female cysts as pearly white, rounded or pyriform bodies, large enough to be seen with the naked eye ($\frac{1}{40}$ to $\frac{1}{25}$ inch).

Some gall development on aerial parts has been recorded, but recent studies have shown that artificial infections may be produced on buds, leaves and stems of susceptible hosts. In the cowpea, leaves infected in the bud may show distortion, crinkling, mild mottling, vein clearing and occasional enations. These results suggest that aerial infections are more common than previously supposed.

Etiology.—Root knot is caused by the minute roundworm, *Heterodera marioni* (Cornu) Goodey, which penetrates the parenchyma of young roots and, by its presence (chemical not mechanical stimulus), induces hyperplasia and also cell enlargement.

The young larvae which enter the tender root tissue in the case of a primary infection develop to sexual maturity within the root, pair and soon the female enlarges and eggs are produced, 300 to 500 in number, extruded in egg masses or oöcysts. The individual eggs are colorless,

transparent, oval-shaped, about $\frac{1}{250}$ inch long and undergo segmentation either within the body of the mother or after being expelled.   The young larva emerges from the egg membrane through a hole which it pierces. Under conditions of high temperature, the egg masses may burst through the side of the root and appear as yellowish, semitransparent bodies closely attached to the root, but, at lower temperatures, the egg masses remain internal.   The young larvae may migrate in the roots with the production of secondary infections, or they may escape into the soil where they may survive for months without any parasitic relation.   In the warmer climates the root-knot nematode may pass through as many as 10 to 12 generations in a year, a life cycle from egg to egg being completed in three weeks to two months.

Environmental Relations.—The root-knot nematode is sensitive to various environmental factors, such as sunlight, cold or heat, drought or moisture and toxic chemicals.   It is generally recognized that climate is one of the limiting factors in the distribution of the root-knot nematode. The amount of root knot below a soil temperature of 60.8°F. is much less than when it is only 2 or 3° higher.   It is almost eliminated 3° lower yet, and, at 50 to 53.6°F., infections are very rare.   Infestations are frequently severe at temperatures

Fig. 187.—Section through a potato invaded by root-knot nematodes. The worms are located in the necrotic areas just beneath the surface. (*After W. A. Orton.*)

up to 85°F. and occur to some extent at much higher temperatures.   The thermal death point for larvae is 128°F., for eggs 137°F., or 10 minutes at 110°F. for larvae and 10 minutes at 119°F. for eggs.   It is the temperature relation that is largely responsible for the present geographic range of root knot as a serious pest.   The temperature requirement of the root-knot nematode makes it possible to grow a low-temperature crop (45 to 60°F.) like lettuce or celery in the greenhouse in a nematode-infested soil which would give heavy infection on tomatoes or cucumbers at 70 to 75°F.

Soil-moisture conditions favorable for crop growth are also favorable for root-knot development.   Root knot can develop to some extent even below 40 per cent soil moisture, which is too low for good crop growth and

above 80 per cent, which is too wet. Flooding has been suggested as a means of killing the soil population with varying results reported. Some practical results have been obtained by flooding truck lands for 3 to 4 weeks. Experimental tests have shown that it required flooding for 4 months to kill larvae and 12 to 23 months to kill eggs, a requirement that would eliminate cropping for two years.

The root-knot nematode is sensitive to drying, the larvae being killed by drying alone in 3 minutes and by drying and sunlight in 2 minutes,

Fia. 188.—Eggs and young root-knot worms just hatched, taken from a potato. (*After F. B. Headley.*)

while the more resistant egg masses require 2 hours of drying only or only 30 minutes if supplemented by direct sunlight. Since nematodes penetrate far below the plow line, desiccation of the soil would not be practical for field culture.

Root knot is likely to be most serious on light, sandy or muck soils, but less serious in clay soils. Some reports have suggested that the disease is favored by acid soils, but definite tests have shown no great difference in the amount of infection in pineapple or cowpeas at a pH range from 4 to 8.5. Some reductions in the soil populations of the root-knot nematode during the decomposition of organic matter have been recorded. Nematode-trapping fungi may reduce the population and of five different species studied, *Dactylella ellipsospora* was proved experimentally to restrain the rise or increase in population. It is the belief that some of the other fungi act in a similar way but to a lesser degree.

· **Host Relations.**—The root-knot nematode attacks an enormous number of plants. Its known hosts included over 855 wild or cultivated species as recorded in 1933, but the most recent compilation includes 1332 (Buhrer, 1938). The following are listed as especially subject to the disease: (1) *field crops*, including alfalfa, clover, cotton, cowpea (exceptions below), sugar beet, sugar cane, sweet potato, tobacco and vetch; (2) *ornamental and drug plants*, represented by begonia, cineraria, clematis, coleus, dahlia, hollyhock, ginseng, goldenseal, peony; rose, sweet pea and violet; (3) *truck crops*, such as asparagus, bean, beet, cantaloupe, carrot, celery, cucumber, dasheen, eggplant, garden pea, lettuce, muskmelon, okra, onion, pepper, potato, · salsify, spinach, strawberry, tomato and watermelon; and (4) *woody hosts*, the most important being the almond, catalpa, cherry, date palm, European elm, fig, mulberry, Old World grapevine, peach, pecan, Persian walnut and weeping willow. A recent compilation (Barrons, 1938) records 30 generic groups, some including one or more species with several varieties that have shown more or less resistance. This suggests that selection of the more resistant varieties offers some hope of reducing the losses. Many wild plants, including most of the common weeds, are also attacked. Nematode attacks may increase susceptibility to certain other diseases, for example, cotton wilt, tobacco black shank and the Rhizoctonia disease of peanuts.

The following list includes the more important cultivated plants which by their immunity or resistance are safe for use in crop rotations on contaminated land: barley, corn, rye, wheat and winter oats; sorghum, milo and kaffir corn; redtop, timothy and nearly all grasses; Iron, Brabham, Monetta and Victor cowpeas; peanut, Laredo soybean, velvet bean and *Crotalaria spectabilis*.

Control.—Many different practices are of value and selection should be made to fit the conditions and the crop:

1. The use ⭕f a catch crop, that is, growing a susceptible crop followed by its destruction before the nematodes migrate into the soil. Tomatoes in pot cultures have removed 98 per cent of the effective population.

2. Selection of a low-temperature crop for infested greenhouse soils, for example, lettuce or celery, or early planting of adapted varieties in the open to ensure maturity before the period of high temperatures.

3. The cultivation and high fertilization of orchards and ornamental plantings, with the avoidance of susceptible cover crops.

4. Complete fallow with the destruction of all weeds, with or without flooding.

5. The use of resistant or immune varieties whenever practical (see Host Relations). Some promising resistant varieties have been found by selection and breeding in the following susceptible crops: tobacco, sweet potato, peach and beans.

6. Crop rotation to include an immune cereal the first year, a very resistant legume the next year, followed by the desired crop. A suggested rotation for the home vegetable garden is: *first* year, chicken runs; *second* year, garden; third year, corn, using three plots or chicken runs so that one section is available for garden each year.

7. Soil sterilization, especially for crops under glass or in intensive field culture. This method is not practical or is too expensive for extensive field culture. The methods include: (*a*) the use of a toxic chemical or nemacide the most important including hydrocyanic acid gas (HCN), carbon bisulphide and "cyanamid" (calcium cyanamid). Methyl bromide, chlorpicrin and ethylene dichloride have shown some promise on the basis of recent tests; (*b*) sterilization with hot water or steam. Crop rotation and soil sterilization are of outstanding importance.

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NEMATODE DISEASE OF WHEAT

Anguillulina tritici (S.) G. and von Ben.

This disease of wheat and other cereals transforms the kernels into galls which have received such names as "smut," "bunt," "hard smut,"

"cockle," "purples," "bin burnt," "immature wheat," "false ergot" or "peppercorns."

The disease was first noted in England in 1743 and now has almost a world-wide distribution, although localized in certain areas. It has not yet appeared in South Africa but has been recorded from North Africa and all of the other continents. In the United States, the disease has been prevalent in 53 counties of West Virginia and to a lesser extent in

FIG. 189.—Malformation of wheat leaves and stems due to the nematode (*Anguillulina tritici*). (*After Leukel, Jour. Agr. Res.* **27**, 1924.)

North Carolina, South Carolina and Georgia but has not reached the principal wheat-growing sections. Because of the localized occurrence only a brief synopsis of the important features will be presented.

Symptoms and Effects.—The following features are characteristic:

1. The wrinkling, rolling, and distortion of the leaves of young plants and the enlargement of stems of affected shoots.

2. Small, raised, rounded areas or galls on some of the leaves.

3. Leaves may become chlorotic, wilt and die in young plants, and, in severe infections, they may become so wrinkled, twisted and rolled as to lose all semblance of their natural form.

4. Older plants may be dwarfed, yellowish, and show some curling of leaves.

5. The heads are shorter, the glumes are more divergent and remain green longer than normal, while the grains are replaced by hard, light-brown or dark-colored galls, smaller and sometimes plumper than normal wheat grains.

The injury from this nematode disease may be caused by: (1) the killing of seedlings, thus reducing the stand; (2) weakened vitality and a

Fig. 190.—Nematode galls and normal grains. *A*, galls from wheat; *B*, galls from rye; *C*, normal wheat kernels; *D*, normal rye kernels. (*After Leukel, Jour. Agr. Res.* **27**, 1924.)

reduced yield; and (3) the lowering of the market grade or quality of the crop. Few to as many as 90 per cent of the seedlings have been killed, and in Virginia the threshed crop has contained from 0.1 to 25 per cent of galls with a maximum of 50 per cent.

Etiology.—This disease is caused by *Anguillulina tritici* (S.) G. and von Ben., one of the parasitic nematodes which may be found in the galls on the young leaves, in the flower or seed galls, and also as an ectoparasite within the leaf sheaths; also free-living for a time in the soil. The parasitism of this nematode has been demonstrated by planting whole or broken galls with normal wheat and also by pouring

an infusion of broken galls over the seed or into the trenches at planting time.

The yellowish-white center of a nematode gall is a mass of these minute roundworms in their *second larval stage*. If a gall is soaked in water and then opened, the freed larvae straighten out and soon begin their characteristic, eellike movements. These larvae are 658 to 910 μ in length and 15 to 20 μ in diameter. Their structure is well shown in Figs. 191 and 192.

Fig. 191.—Lateral view of young female of *Anguillulina tritici* (for explanation of letters see Fig. 192). (*After Byars, U. S. Dept. Agr., Bul.* **842,** 1920.)

The larvae from galls falling to the ground or mingled with the seed grain escape into the surrounding soil. If they come in contact with susceptible seedlings, they penetrate between the leaf sheaths near the apical or growing points of the culms and remain in this position until the head is produced, or they cause leaf galls or give rise to the characteristic curled and rolled leaves. When the heads are formed, some of the larvae enter the flowering parts and, within the ovaries or adjacent organs, become sexually mature, pair, and lay thousands of eggs. The old worms die, the eggs hatch, and the newly formed larvae become dormant by the time the galls have reached maturity.

Sexually mature males and females develop within the galls, the latter reaching a length of 3.5 to 4 millimeters and 168 μ or more in width. Most of the body back of the oesophagus (see Fig. 191) is occupied by the egg-producing organs, consisting of a short posterior sterile portion and a large anterior fertile portion. A single female may lay more than 2000 eggs. The males are smaller than the females, 2 to 2.5 millimeters long and more slender (see Fig. 192 for parts).

The eggs are elongated ellipsoid, 73 to 140 by 33 to 66 μ and covered with a tough transparent skin. Segmentation proceeds rapidly after the eggs are laid and soon the young larva pierces the egg membrane with its buccal spear and escapes from the shell. The larvae pass into a dormant or very resistant state by the time the galls are mature: In this condition they may remain dormant for many years and resume their life cycle when favorable conditions are offered. The dormant larvae

in the galls are very resistant to temperature changes and to chemical agents, being more resistant in the dry galls than when moist.

Host Relations.—While wheat is the principal host, other cereals may become infected. Spelt has been noted as a host under natural field conditions, while emmer, rye, and spelt have shown abundant infections and oats and barley light infections by artificial inoculations. Flower galls due to similar nematodes have been observed on various wild grasses, but these nematodes will not infect wheat, and the wheat nematodes will not infect any of the wild grasses. The flower-infesting nematodes of the wild grasses are either a distinct species or biological strains of the wheat nematode.

Control.—The disease can be reduced to a minimum by the use of clean or nematode-free seed wheat or by the rotation of crops.

Clean seed may be obtained: (1) by the selection of seed from localities known to be free from the disease; (2) by the separation of the galls from contaminated seed; and (3) by the hot-water treatment to kill the nematodes. The galls may be separated by the salt-brine treatment, in which the contaminated seed is poured into a 20 per cent solution of common salt (40 pounds to 25 gallons) and stirred vigorously to bring the galls to the surface. The floating material is then decanted into a second container covered with a cheesecloth screen, and the clean wheat rinsed in pure water and spread out to dry. As an extra safeguard, the cleaned wheat may be treated with water at a temperature of 51 to 52°C. for 10 minutes.

Fig. 192.—Ventral view of young male of *Anguillulina tritici*, *A*, spear; *B*, anterior oesophageal bulb; *C*, oesophageal canal; *D*, posterior oesophageal bulb; *E*, digestive system; *F*, reproductive system; *G*, spicula in the male and vulva in the female; *H*, anus; *I*, bursa of male; *J*, tail. (*After Byars, U. S. Dept. Agr. Bul.* **842**, 1920.)

Hot-water treatment alone is effective. Soak the grain 1 hour in unheated water, immerse immediately in hot water using any one of the following combinations: 20 minutes at 52°C.; 15 minutes at 54°C. or 10 minutes at 56°C.

Crop rotation must be practiced if the previous crop of wheat was infected. A nonsusceptible crop should be grown for two consecutive years or better for three years, using preferably a cultivated crop for the first year. Good results have been obtained, however, by using clover, alfalfa or other legume one or two years, followed by corn for one year, before returning to wheat.

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IMPORTANT DISEASES DUE TO NEMATODES

For key references to these diseases see F. D. Heald, "Manual of Plant Diseases," 2d ed., pp. 898–899.

Principal host	Common name of disease	Scientific name of causal organism
Wheat, also other cereals and some grasses	Nematode disease	*Anguillulina tritici* (S.) G. and v. Ben.
Numerous (220+)	Stem and bulb nematode	*A. dipsaci* (Kühn) G. and v. Ben.
Oats	Tulip root	*A. dipsaci* (Kühn) G. and v. Ben.
Rye	Stem disease	*A. dipsaci* (Kühn) G. and v. Ben.
Clover and alfalfa	Stem sickness	*A. dipsaci* (Kühn) G. and v. Ben.
Potato	Eelworm disease	*A. dipsaci* (Kühn) G. and v. Ben.
Onion	Bloatiness	*A. dipsaci* (Kühn) G. and v. Ben.
Hyacinth and narcissus	Eelworm or ring disease	*A. dipsaci* (Kühn) G. and v. Ben.
Phlox	Eelworm disease	*A. dipsaci* (Kühn) G. and v. Ben.
Rice	Rice nematode	*A. angusta* (Butler) Goodey
Various grasses (12)	Grass nematode	*A. radicicola* (Greef) Goodey
Banana, sugar cane and others (18)	Burrowing nematode	*A. similis* (Cobb) Goodey
Numerous (50+)	Root nematode	*A. pratensis* (deMan) Goffart
Citrus	Citrus-root nematode	*Tylenchulus semipenetrans* Cobb
Beets, oats, peas, potatoes, etc. (90+)	Beet nematode	*Heterodera schachtii* Schmidt
Numerous (800+)	Root knot	*H. marioni* (Cornu) Goodey
Strawberry	Strawberry nematode Cauliflower disease Red plant Dwarf or crimp	*Aphelenchoides fragariae* (R. Bos) Goodey
Chrysanthemum and 20 other ornamentals	Chrysanthemum nematode	*A. ritzema-bosi* (Sch.) Goodey
Ferns (50+) and flowering plants (50+)	Fern nematode	*A. olesistus* (R. Bos) Goodey
Coconut and date and oil palms	Palm nematode	*A. cocophilus* (Cobb) Goodey

SECTION III
VIROUS DISEASES

CHAPTER XVI

TYPES OF VIROSES AND GENERAL NATURE OF VIRUSES

The virous diseases of plants constitute one of the great groups of plant troubles which stand out rather distinctly from those due to non-parasitic or environmental factors as well as from those in which a visible parasite is the causal agent. The virous troubles have one feature in common, namely, they may be transmitted from diseased to healthy plants by an infectious principle—the virus. Certain conditions of common occurrence in plants, like variegation or noninfectious chlorosis, seem to be very closely related to the virous troubles and may well be considered at the beginning.

Noninfectious Chlorosis.—In noninfectious chlorosis, there may be a more or less uniform yellowing of the foliage, or the leaves may show areas of white or yellow mingled with regions of normal green, giving the type of chlorosis that is called "variegation." Several different types of variegation may be recognized: (1) marginal variegation, with narrow or broad zones of pale tissue marking the edge of leaves or leaflets; (2) sectional variegation, with the yellow and green areas distributed over the leaves and stems or leaves only, in the form of blotches, spots, bands or stripes; and (3) marbled and pulverulent variegation.

When genuine chlorosis or variegation appears in food plants, it is considered to be a detrimental or diseased condition since the photosynthetic power of the plant is modified or reduced in accordance with the degree or intensity of the yellowing. When, however, these peculiarities of the foliage appear on ornamental plants, they are considered as adding to their decorative value, and the variegated forms are preserved. These abnormal or chlorotic forms appear in the trade as horticultural varieties of the normal species or variety from which they originated, under such names as *aurea, variegata, alba, argentea, aureo-, albo-* or *argenteo-marginatis,* etc. This type of chlorosis may be illustrated among herbaceous forms by ribbon grass (Phalaris), zebra grass (Miscanthus), variegated periwinkle (Vinca) and variegated varieties of the common garden nasturtium (Tropaeolum); among shrubs by the golden elderberry, a form with golden-yellow or yellowish-green leaves, and many

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variegated forms such as the variegated elderberry (*Sambucus spp.*),
burning bush (Euonymus) and althea (Hibiscus); and among trees, by
variegated holly (Ilex), the golden-leaved English elm, the variegated
maples and the golden box elder.

Variegation may appear spontaneously in almost any herbaceous or
woody species either growing wild or under cultivation, without the
operation of any known inciting factors.   In food plants, these variega-
tions are either ignored or eliminated by selection; while, in ornamental
plants, they may be propagated.   In the great majority of cases, variega-
tion can be propagated only by the use of buds or cuttings, which usually
come true to type, although in some cases a certain percentage may
revert to the normal green form.   The majority of variegated forms do
not come true from seed, but exceptions may be noted, as in the variegated
nasturtium, in *Ptelea trifoliata aurea*, etc.   Variegated forms generally
appear quite healthy, but it has been noted that they are not so hardy
as their normal green progenitors.   They are reported to succumb more
readily to unfavorable conditions, such as drought or cold and in some
cases to be more susceptible to the inroads of parasitic forms.

Types of Virous Diseases.—No satisfactory classification and
nomenclature of the virous diseases has yet been presented.   It has been
customary to apply names on the basis of hosts attacked or of symptoms,
but such a classification is inadequate.   If a virus is a distinct chemical
entity, or even as distinct as, and no more variable than, a bacterial
or fungous entity, a logical classification may be forthcoming.   Recent
discussions on classifications have been published by Johnson and Hogan
(1935), Kunkel (1935), Birkeland 1936), Holmes (1939), Ainsworth (1939),
Fawcett (1940) and Thornberry (1941).   A detailed treatment of these is
beyond the scope of this discussion, but no one is entirely satisfactory.
That of Holmes is most complete and has been adopted by some authors.
Recent general handbooks or texts have been issued dealing with phyto-
pathogenic viruses as follows: Grainger (1934), Smith (1937 and 1940),
Holmes (1939) and Bawden (1939).   On the basis of present informa-
tion, certain groups may be recognized which represent single virous
entities, related or similar viruses or groups of viruses.   With present
information, it is not possible to make a certain assignment of some
of the recognized diseases.   Some of the more important groups are as
follows:

1. Infectious chlorosis, general chlorosis or variegation transmitted
only by organic union as in budding or grafting but not contagious.

2. The peach group including yellows, little peach, peach rosette,
red suture, phony peach and peach mosaic, all transmitted by organic
union but only peach yellows by a known insect vector, *Macropsis
trimaculata*.

3. Wheat mosaic and rosette transmissible to all species of the tribe Hordeae and in nature communicated in some way through the soil.

4. Curly top of beets and many other cultivated crops as well as numerous wild plants. In nature, transmitted by the leaf hoppers, *Eutettix tenellus* and *Agallia sticticollis*.

5. Aster yellows transmitted by the leaf hopper, *Cicadula sexnotata*, to numerous hosts, including aster, celery, lettuce, etc.

6. Potato mosaics, including several distinct viruses or virous complexes, transmitted mainly by aphids.

7. Potato leaf roll, a single virus entity transmissible to some other Solanaceae.

8. Tobacco mosaics, including a group of distinct or closely related viruses.

9. Tobacco ring spot transmissible to other Solanaceae and many other unrelated species in 16 different families.

10. Bramble viroses, caused by a group of virous entities, affecting raspberry, blackberry and loganberry.

11. Strawberry viroses, caused by several apparently distinct virous entities.

12. Cucumber or cucurbit mosaics, including a group of distinct viruses affecting various cucurbits and also some species from other families.

13. Legume mosaics, including a number of distinct or closely related viruses, many transmissible by the seed.

14. Hop viroses, including several viruses, apparently confined to the nettle family.

15. Bulb mosaics or other viroses, including a number of viruses not yet well differentiated.

16. Bunchy top, affecting banana, plantain and Manila hemp, probably caused by a single virus.

17. Grass mosaics, affecting various wild or cultivated grasses, sugar cane and corn, probably includes several distinct viruses.

18. Grass streaks, affecting corn, sugar cane and various other Gramineae, probably including several either distinct kinds or strains.

In addition to the viruses included in the groups enumerated, the following appear to represent distinct viruses: cotton leaf curl, reversion or nettlehead of currants, cranberry false blossom, potato spindle tuber, spotted wilt of tomato, curl disease of beet, spike disease of sandal and pineapple yellow spot. There are other less known virous diseases in which the virus may prove to be either distinct or referable to some of the recognized entities.

Certain virous diseases have been shown to represent double infections, that is, the combined action of at least two distinct viruses, for example,

the streak of tomato due to the X (latent) virus of healthy potato combined with tobacco mosaic. Potato X virus in combination with Y virus appears to give rugose mosaic symptoms on potato, and other mixtures are known. It seems probable that other viruses now considered to be caused by single virous entities will be resolved into two or even more separate entities as methods of separation and purification become perfected.

The Infectious Nature of Virous Diseases.—The mosaic disease of tobacco was the first virous disease that was proved to be transmissible from diseased to healthy plants. The discovery was made by Iwanowski (1892) that extracted juice of a tobacco plant affected with mosaic would infect a healthy plant if pricked into its tissues, even though it had been passed through a Chamberland filter. Similar results were later obtained by Beijerinck (1898), and the filterable character of the infective principle of tobacco mosaic and other viroses has since been repeatedly demonstrated. The first record of insect transmission of a virous disease is in the work of Takami (1902), who connected the "stunt" disease of rice with the leaf hopper, *Nephotettix apicalis.* The beet leaf hopper (*Eutettix tenellus*) was proved by Ball (1906–1909) and Shaw (1910) to be the vector for beet curly top (see this disease). The transmission of tobacco mosaic by aphids was first demonstrated by Allard (1914). The connection of aphids with potato leaf roll was pointed out by Botjes (1920) and confirmed a few years later by Schultz and Folsom (1923, 1925) and Murphy (1923). Since that time, rapid progress has been made in the determination of the insect vectors of the various viroses.

Methods of Transmission.—Virous diseases show different degrees of infectiousness and consequently may be transmitted in different ways in nature or artificially as follows:

1. *Budding or Grafting.*—This includes all viroses but some are transmitted in no other way, including infectious chloroses, all peach viroses except yellows, potato witches'-broom, hop mosaic, spike disease of sandal, etc. Insect vectors for these may exist, but they have not yet been discovered.

2. *Inoculation.*—As related to a virus this means the actual application of the virus-containing sap to the suscept. This method is successful in most of the mosaics and in some other viroses.

3. *Seed* (True Seed).—Most viroses are not carried by the seed produced by infected plants. Seed transmission is certain for many legume mosaics, lettuce mosaic, two ring spots of tobacco, petunia mosaic and the mosaic of wild cucumber and is suspected in some other cases.

4. *Contact Transmission.*—The extreme sensitiveness is illustrated by tobacco mosaic, which may be transmitted by contact or by touching a diseased plant and then touching a healthy one. It has been proved

that workmen who chew tobacco can introduce the mosaic by wiping the mouth and then weeding and handling the plants or even by "spitting in the bed." A similar transmission of the tobacco-mosaic element of tomato streak by tobacco chewers has been responsible for the ruination of tomatoes under glass. Several other cases of apparent contact transmission have been reported including root mutilation in narcissus stripe, by simple contact from diseased to healthy plants in rugose mosaic, and possibly in wheat mosaic although these cases appear to need further confirmation.

5. *Pollen.*—Mosaic of Jimson weed, and bean mosaic are the only reported cases of pollen transfer, but it seems probable that other cases will be proved as this feature is tested.

6. *Insects.*—A very high percentage of our virous diseases are known to be transmitted by the feeding punctures of insects, and many others for which the vectors have not been discovered are thought to be insect-borne. It is probable that no insect vectors exist for some viroses, for example, infectious chlorosis, and X virus of potato.

7. *Parasitic Seed Plants.*—Dodder (*Cuscuta campestris*) may act as a vector by growing from a virus-infected plant to one that is virus-free. This method has been demonstrated for six different viruses (Johnson, 1941).

Insect Vectors of Viroses.—The insects responsible for the transmission of virous diseases may be divided into two groups: (1) *Mouth parts adapted for biting.* These are relatively unimportant, only a few authentic cases being recorded: cowpea mosaic by the bean leaf beetle, cucumber mosaic by the two cucumber beetles and potato spindle tuber by grasshoppers and a few beetles. In the case of biting insects, there seems to be no specificity in the transmission, the transfer or inoculation being mechanical. (2) *Mouth parts adapted for sucking.* The principal vectors belonging to this group are: (*a*) Thrips, for example, *Frankliniella insularis*, the chief vector of spotted wilt of tomato and *Thrips tabaci*, connected with the transmission of several different diseases, and several other doubtful cases. (*b*) Plant bugs, leaf hoppers, white flies, aphids and coccids. Examples: Tingia bug, *Piesma quadratus* and leaf curl or crinkle of sugar beet; leaf hoppers, *Eutettix tenellus* and curly top of beet and other hosts; *Cicadula sexnotata* and aster yellows, and at least four other leaf hoppers as vectors of streak of maize, cranberry false blossom, mosaic of rice, and peach yellows; white flies, *Bemisia gossypiperda* and leaf crinkle of cotton in the Sudan; aphids or plant lice, at least 23 species as the vectors of 27 or more viroses on numerous plants; and a few cases of coccid transmission.

Some of the more important factors in the relationship between viruses and insect vectors may be briefly enumerated: (1) numerous cases are

on record of a delay in the development of the infective power (a period of incubation) following feeding upon a diseased plant; (2) there is an apparent specificity of certain insects for particular viruses, as illustrated by diseases in which only a single species of vector is known or by those in which certain vectors are much more efficient than others; (3) no morphological or cytological differences between viruliferous and noninfective insects have been discovered; (4) many vectors retain their infective power for a long time or even during their whole lifetime, indicating the possibility of an increase of the virus within the body of the insect; and (5) the virus is not transmitted by young from the eggs of viruliferous parents until after they have fed upon infected plants. The evidence appears to indicate that the relationship between vector and virus is something besides a mere mechanical connection.

It is also of interest to note some of the relationships between the plant and the insect vectors: (1) there may be a selective transmission; that is, a certain insect may transmit only one virus after feeding on a plant containing a mixture; (2) the virus exists in all parts of a plant, but it cannot be obtained by a vector from all parts with equal ease; (3) a virus may exist in a plant without causing any external evidence of its presence, and such symptomless "carriers" may yield the virus to insects feeding upon them; and (4) infection may result from the feeding of a single viruliferous insect, but infection is greater with large numbers of vectors. Three general groups of transmission types may be recognized: (1) external transmission by the carriage of the virus on the mouth parts of the feeding insect; (2) mechanical internal transmission involving no development or multiplication in the body of the vector; and (3) biological internal transmission in which the virus increases in the body of the vector.

Symptoms and Effects of Viroses.—The general effects of viroses may be grouped under three divisions: (1) a general chlorosis, a vein clearing or a localized mottle or mosaic of the foliage; (2) a necrosis of tissue causing dead brown spots, streaks or stripes and, in the extreme, a killing of extensive areas or whole organs; and (3) dwarfing, distortion of growth or malformations involving single organs or the entire plant. A single virus at different stages of the infection or upon other hosts may be responsible for abnormalities in all of these groups.

A general paling of leaves and other green parts without the appearance of any mottle or mosaic with the final production of a rather *general chlorosis or yellowing* is a very pronounced symptom in certain troubles and is well illustrated in aster and other suscepts affected by the aster-yellows virus, peach yellows, raspberry yellows (curl), tomato yellows or western blight and others.

A primary symptom of certain viroses is a *clearing of the veins,* that is, the tissue adjacent to the veins is paler than the intervening tissue.

This is the only symptom produced on some suscepts, but, in the majority of cases, it is a primary or initial symptom that is followed by the appearance of other more evident effects. It is a noticeable feature in the *Y* virus, aster yellows, curly top of sugar beet and in many of the tobacco mosaics.

Fig. 193.—*A*, marrow bean showing speckled type of mottling; *B*, potato mosaic; *C*, leaf of pea bean showing blistered type; *D*, mosaic of Windsor bean. (*After B. T. Dickson, Macdonald Coll. Tech. Bul.* **2.**)

The mottling of the foliage due to alternating patches or spots of light green or yellow and dark green is characteristic of a very large number of viroses known as *mosaics*. The mosaic mottle may be very mild or only evident as slightly paler green spots or it may be very evident with the extreme represented by distinctly yellow or even white spots. The spots vary in form and size from irregular to angular and from small to large, certain patterns being characteristic of specific mosaics (see Fig. 193). In severe types of mosaic the leaves may be savoyed, or show blisterlike elevations with intervening depressions, the latter occupied by the chlorotic areas when mottling is evident, thus giving the leaf a puckered, wrinkled or rugose surface. This feature is characteristic of

rugose mosaic of the potato and in curly dwarf types without any mosaic mottle. Because of the morphology of flower structures, mosaic effects should be expected on perianth or fruits. Mottling of flowers of sweet peas, petunias and tobacco is caused by mosaics on these suscepts, while breaking of tulips or the production of variegated flowers by solid color varieties like the Darwins, represented by such commercial varieties as Rembrandt and Bybloemen, is also a mosaic effect. Cucumbers affected with mosaic may show a fruit mottle which spreads over the entire fruit, and the dark green areas are usually elevated above the yellow spots giving the fruit a warty surface. Under certain conditions, especially in the greenhouse, the mosaic mottle may develop without the irregular topography, while in late severe stages the fruits may be smooth and pale whitish green, the "white pickle" of commercial growers.

The formation of either complete or broken concentric, chlorotic or necrotic rings of varying size, frequently with necrotic center spot, is a characteristic foliage response to certain viruses. This is well illustrated by the ring-spot disease of tobacco, some infections by the X virus of potato on tobacco, the virus of spotted wilt of tomato especially on other hosts than tomato, the ring spot of peony and a ring-spot symptom as one of the phases of lupine mosaic. The rings may vary in size from a few millimeters to several centimeters in diameter, varying with the particular suscept and virus concerned.

Necrosis or the browning and death of tissues is a marked effect of certain viruses and may involve localized groups of cells, either superficial or internal. The resulting external picture may be necrotic spots, streaks or extended necrotic areas finally involving entire leaves or portions of stems. Internal necrosis may involve the vascular tissue, especially the sieve tubes and companion cells (see Potato Viroses). Necroses are not confined to the vegetative organs but may involve the fruits as may be illustrated by the localized spotting or internal necrosis of tomatoes. Bitter pit of apple has recently been assigned to the virous group, but there still seems some uncertainty as to its virous origin. Killing of roots is one of the effects of curly top on tomatoes and has also been reported for the spike disease of sandal. In severe potato viroses like rugose mosaic, root necroses appear to keep pace with foliage killing. As more attention is given to the root system of virus-infected plants, more and more cases of local or extended root necrosis will be recorded.

Reduction in size of leaves or other organs or of the entire plant is a feature of many of the virous diseases. Stunting with more or less rosetting is characteristic of bunchy top of bananas, stunt of rice, rosette of peanuts, rosette of peach, peach yellows, curly top of beets and others. Reduced size has suggested the common names of several virous diseases, for example, *dwarf* of alfalfa, dwarf of bramble fruits, dahlia stunt, yellow

dwarf of onion, curly dwarf of potato and little peach. Increased pro-liferation is a symptom that is found in various viroses and in extreme cases may produce typical witches'-brooms as in potato, ocean spray, strawberry and alfalfa and by brooming disease of black locust and spike disease of sandal. The extreme development of the witches'-broom in alfalfa has produced plants with as many as 300 stems.

Fig. 194.—Virous disease of lupine showing ring-spot symptom. (*Photograph by L. K. Jones and F. D. Heald.*)

A rolling or curling of the leaves is a frequent symptom in many of the viroses, and this character when especially prominent has frequently suggested the name applied to the disease, as may be illustrated by curly top of beet and other suscepts, cotton leaf curl, leaf roll of grape, leaf curl of poinsettia, potato leaf roll, leaf-rolling mosaic of potato, leaf curls of raspberry, and tobacco curl. Distortion or malformation of leaves may be another response of leaves to a virosis, such as asymmetric development of the laminae, dissection of the margin or reduction of the

leaves to little more than midribs. The extreme types are illustrated by "fern leaf" or "filiform leaf" of tomato produced by cucumber mosaic, and also by the delphinium stunt virus; "filiform leaf" of sweet potato; and the long, sinuous or ribbonlike leaves of tobacco.

The stimulatory effect of a virous infection may be illustrated by the alloiophylly of the anemone with thickened and misshapen leaves and frequently with thickened stems and leaf stalks; by the Fiji disease of sugar cane in which elongated galls are produced on stems and leaves; by conspicuous vein swellings on the underside of the leaves in curly top of sugar beet; and possibly by the giant hill of the potato.

Pathological Histology of Mosaic Plants.—Space will not permit any general discussion of this feature for all types of viroses, and the statement will be limited to some of the more important tissue changes in mosaic foliage as follows:

(1) A reduction in the leaf thickness of the chlorotic areas due to hypoplasia of both spongy and palisade tissue, but especially the latter. The general ratio of thickness between the light-green and dark-green areas of leaves is 2:3. In the extreme cases, the palisade layer may be reduced to a single row of nearly cubical cells, but there are all gradations from this condition to nearly normal. (2) A more uniform and compact arrangement of the mesophyll cells in the light-green areas, with less intercellular space. This makes the light-green areas more transparent than the darker areas. (3) In the light-green areas the chloroplasts may be paler than normal but in usual numbers, while in more severe infections they may be reduced in number and in size with much diminution in color. In very acute infections, the chloroplasts may coalesce into irregular green masses, or they may become completely disintegrated into small, hyaline bodies. This disintegration of the cell contents may produce the dead flecks or spots which are especially noticeable under conditions of prolonged or severe drought. (4) The cells of the dark-green areas are larger than normal, contain more chloroplasts and the chlorophyll is also darker than in normal or healthy leaves. When savoying is pronounced, the palisade cells have either divided to form two layers or they are longer, narrower and more numerous or both. (5) The volume of intercellular space in the dark-green areas is increased, and this feature, together with the greater depth of color of the chlorophyll, makes these areas look darker than normal (see Fig. 204).

The Nature of the Causal Agency in Virous Diseases.—The numerous studies which have been carried out since the recognition of mosaics and related diseases have failed to give positive evidence as to the real nature of the causal agency. An infectious disease, without any visible causal organism, and transmitted by an agent or principle so small that it will pass through ordinary bacteria-proof filters, presents a field of investiga-

tion that is taxing the ingenuity and imagination of scientists. Speculation and theorizing have marked the progress of our knowledge of the virous diseases; but as time has passed, more careful attention to corroborating facts has been given. Of the various possible views as to the causal agency, many have been exploited and discarded, but the four following theories are given attention in current literature:

1. *The Bacterial Theory.*—The fact that various virous diseases behave much like known bacterial diseases gave the earlier workers a prejudiced outlook, and they naturally expected to find bacteria in plants affected with mosaics or other viroses. Such findings were reported by a number of investigators. The careful technique of recent investigators has practically excluded bacteria as possible causal agents. It has been shown that bacteria may sometimes be quite generally associated with virous lesions but that the disease is reproduced by bacteriologically sterile, virus-holding juice, without the presence of any bacteria.

2. *The Enzyme Theory.*—This view was first proposed by Woods in 1899 for tobacco mosaic. He attributed the disease to the extensive development of oxidizing enzymes. With various ideas as to the real nature of the enzymes, this theory was supported by later investigators by comparison with the behavior of known enzymes. This theory involved the assumption that a small quantity of enzyme introduced into normal cells must have the power to start the production of this same enzyme and that this operation once started could not be stopped. The enzyme theory is no longer accepted even by its original proponents.

3. *The Protozoan Theory.*—Amoeboid bodies were first described by Iwanowski (1903) as occurring in the cells of mosaic tobacco plants and later studies by numerous investigators showed that similar plasmalike bodies are to be found in the tissues of many animals and plants affected with virous diseases. These inclusions have been called X bodies, but the present opinion is that they do not represent stages in the life cycle of a causal organism but are rather the results of derangements in the cells induced by the activity of the viruses. It is of interest to note that three different investigators have been sufficiently convinced of the protozoan or organism nature of these intracellular bodies to apply definite binomials to them. Structures which Nelson (1922) identified as typical trypanosomes from the sieve tubes of tomato plants affected with mosaic and potato plants showing leaf curl were shown by other workers to be normal inclusions in healthy as well as diseased cells.

4. *The Filterable-virus Theory.*—The term "virus" has been used without any preconceived concept as to its nature, for the filterable agent or principle which has the power of inducing disease. Various ideas have been held as to the nature of the virus of which the more important of current interest are: (*a*) A noncorpuscular, water-soluble

substance, the *contagium vivum fluidum* (Beijerinck, 1899). (*b*) The "invisible microorganism" concept which pictured the virus as a minute, possibly bacterialike organism, so small as to be beyond the range of microscopic vision. At first the ability of the virus to pass through the pores of certain standard bacteria-excluding filters was not opposed to the microorganism concept, but later tests showing the minuteness of the infective particles (estimates range from 5.5 to 30 millimicrons) have made the theory a difficult one to accept. The virus particles have been shown to differ in shape as well as size, some being nearly spherical, others rod-shaped, and still others asymmetric in form. (*c*) A chemical entity, the exact nature not specified, but a product of host cells, which has "revolted from the shackles of coordination and, being endowed with the capacity to reproduce itself, continues to produce disturbance and 'stimulation' in its path, but its path is only the living cell." (Duggar and Karrer, 1921.) This theory was based on circumstantial evidence rather than on any isolation of the virus. (*d*) A highly infectious crystalline protein (Stanley, 1935, and later; Best, 1939). This is based on the isolation and purification of a crystalline protein containing 20 per cent nitrogen and 1 per cent ash from tobacco mosaic on both tobacco and tomato which is 100 times more active than a suspension prepared by grinding up diseased tobacco. This material has been recrystallized repeatedly with the retention of constant physical, chemical and biological properties, and is believed to be a definite pure chemical compound and, therefore, inanimate. This virous protein may be of the autocatalytic type requiring the presence of living cells for its multiplication. The occurrence of strains of tobacco mosaic virus may then be explained on the chemical basis, with changes in the protein molecule to give similar or isomeric proteins. Possibly this may explain the reported mutation of viruses. If there is any weak link in the chain of evidence, it is the fact that the virosis studied is so highly infectious that the possibility exists that the active principle may still be held within the protein isolated. Similar work with some of the viroses more difficult to transmit appear to be necessary to substantiate the theory.

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CHAPTER XVII

DISEASES DUE TO VIRUSES

INFECTIOUS CHLOROSES

The chloroses of plants that are not the result of environmental factors may be (1) variegations or noninfectious chloroses perpetuated either by seed or by vegetative propagation; (2) chloroses transmitted only by graft union of chlorotic and green parts, the "infectious chloroses"; and (3) infectious chlorotic conditions or "mosaics" transmitted by juice or insects.

Symptoms.—The amount of variegation and the exact patterns vary in the different species or even in the same species under different influences. The variegation may vary from such forms as *Abutilon indicum,* showing "a single more or less expanded yellow spot in the leaf with little of the green remaining, to others with only a slight yellowing along veins or at the tips of marginal leaf teeth as in *Sorbus aucuparia;* or the leaves may show large or small yellow spots with a typical mosaiclike character in certain cases." In some cases, affected leaves may remain smaller than normal ones and show some wrinkling or rugosity. In cases showing the most extreme reduction of the chlorophyll-bearing surface, the plants may be killed because of inhibited photosynthesis.

Etiology.—The chlorosis or variegation of the foliage in this group may be transmitted from chlorotic stock to normal green stock by budding or grafting or even in some cases by transplanting a piece of diseased cortex in the cortex of a normal plant. In general, actual organic union between chlorotic and normal tissues appears to be necessary for the transmission of the chlorotic disturbances. The establishment of the graft upon the scion appears to be the important feature. All kinds of inoculations using juice, filtered or unfiltered, or macerated or ground tissue, also insect transfer, have failed.

The following data are based largely on the contributions by Baur (1906–1908):

There are no external characters which indicate whether a given form is infectious or will behave as ordinary variegation. For example, of three variegated varieties of privet (*Ligustrum vulgare*)—(1) *albomarginatis,* with white leaf margins; (2) *aureum,* a typical golden form; and (3) *aureovariegatis,* with yellow spotted leaves—only the last proved to be infectious, while the two others were of the noninfectious type.

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It has been shown that the virus or the infective principle is produced in the variegated leaves under the action of direct sunlight. If the old variegated leaves of an abutilon or any other species showing infectious chlorosis are darkened, the new leaves that appear at the growing point will be pure green even though exposed to the light, but darkening of the growing point alone does not prevent the variegation in the newly formed leaves. Partial darkness seemed to have the same effect upon the production of the virus as complete darkness.

If *Abutilon thompsonii*, a variegated form, and a normal green *A. indicum* are grafted on adjacent branches of *A. arboreum*, a species immune to infectious chlorosis, the new shoots of *A. indicum* will be chlorotic, indicating that the infective principle was transmitted through the tissues of the immune stock. In similar tests with an immune Lavatera, the virus was not transmitted. In no cases of infectious chlorosis is the virus transmitted to the embryo plant in the seed.

It has been pointed out that both infectious and noninfectious chloroses may be associated in the same plant. In *Euonymus japonica*, for example, infectious chlorosis may be associated with other noninfectious variegations of the "marmor," "chlorinomarginata" and "aureomaculata" types, thus masking or obscuring the infectious type. Separation of the two can be accomplished by grafting the variegated form on one having normal, uniformly green leaves.

Observations and transmission experiments have demonstrated two distinct types of infectious chlorosis in the mallows: (1) in *Abutilon striatum thompsonii*, showing as a yellow spotting of the leaves and a yellow coloration of the veins; (2) in *A. darwinii tesselatum*, characterized by pale green spots and stripes on the leaves, but with the veins green. *Lavatera arborea* is immune to the (1) type but very susceptible to the (2) form, which causes complete deforming of the leaves, followed by death, while other Malvaceae are susceptible to both types.

Hosts.—Variegated varieties that represent infectious chloroses may be found in the following families and genera: Malvaceae (*Abutilon spp.*); Celastraceae (*Euonymus japonica*, the Japanese burning bush); Oleaceae (*Ligustrum spp.* or privets and *Jasminum spp.*); Leguminosae (*Laburnum vulgare* or Golden Chain); Cornaceae (*Cornus alba*, or Tartarian dogwood); Rutaceae (*Ptelea trifoliata*, or hop tree); and Rosaceae (*Sorbus aucuparia* or European mountain ash).

<div align="center">References (H. 265)</div>

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PEACH VIROSES

YELLOWS

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Peach yellows occurs in the area from Massachusetts south to the Carolinas, the southern boundary including most of Tennessee and crossing Arkansas and Oklahoma, the northern boundary crossing New York,

the .peach districts of Ontario and Michigan and the western range extending into Missouri and Kansas. It does not occur west of the Rocky Mountains and is confined to America.

The disease may be recognized by the following: (1) *Fruit characters.* Premature ripening, frequently abnormally enlarged, more watery than normal, insipid flavor, and often speckled or blotched with red, with the

Fig. 195.—Peach yellows. *A*, typical "willow" shoot showing the upright, much-branched habit with small, yellow foliage, with normal leaf for comparison; *B*, terminals showing slender growth, narrow leaves, upright habit; *C*, healthy shoot for comparison. (*After W. A. McCubbin, Pa. Dept. Agr. Bul. 382.*)

color confined to the skin or with internal spots and streaks. (2) "*Willow*" or "*broom*" *shoots.* ,Slender, much branched, more or less wiry shoots, clothed with small yellow leaves, often spotted with red. They are more slender than water sprouts, grow erect rather than spreading, and may be lateral or terminal. Leaf and blossom buds start growth earlier in the spring and willow shoots frequently make a later fall growth. (3) *Foliage changes.* The shoot axes are short, the leaves generally pale green or yellow, reduced in size, being narrower than normal, more or less drooping and rolled or curled to make them more or less tubular.

Infected tissues contain intracellular inclusions shaped like tadpoles and measuring up to 3 μ. These appear to be located in the cell sap of the vacuoles (Hartzell, 1937).

Affected trees may die the second year after the first evident symptoms appear or the progress of the disease may be slow, the branches succumbing from the top downward. In these slower cases the affected trees may survive in a crippled condition as long as many apparently normal trees. The disease causes heavy losses as a result of death of trees and shortening of the life of the orchard, and the immediate removal and destruction of trees is necessary even before they have come into bearing. The annual losses during even quiescent periods may range from 1 to 3 per cent, while during epiphytotics the removals may reach to 25 per cent.

Early studies of the disease proved that it could be transmitted to healthy trees by budding, and that the smallest amount of bud tissue that could be used and form a union was sufficient to communicate the disease, but the method of transmission under natural conditions was not determined until the work of Kunkel (1933). Previous to this time, it had been shown that the disease could not be transmitted by pruning tools, by pollen from diseased trees, or by mechanical juice inoculations from diseased to healthy trees, but the behavior of the disease pointed to some undiscovered insect vector. It has now been established with certainty that peach yellows is transmitted from diseased to healthy trees by the feeding punctures of the leaf hopper, *Macropsis trimaculata*, an insect which is prevalent in the regions in which peach yellows occurs, although not in such conspicuous numbers as in the case of some other known vectors of virous diseases. The disease has been experimentally transferred by both nymphs and adults, and it has been shown that they feed primarily upon young twigs rather than on the leaves. The delayed discovery of the vector can be explained by the paucity of population, and the fact that they seldom fly, but run rapidly and retreat to the opposite side of a limb from the observer. The vector is much more common on the native wild plum (*Prunus americana*) than on the peach, and undoubtedly this species serves as a center from which viruliferous hoppers spread to adjacent peach trees.

Previous to the discovery of the insect vector, the immediate removal and destruction of the diseased trees had been a recognized practice, with constant watch for the first evidences of infection. In some states, regular state inspection of all commercial orchards has been in operation to supplement the efforts of the owners. With the new information as to the habits of the vector, it would seem that the removal of wild plums from the vicinity of peach orchards would aid in the control of this disease.

LITTLE PEACH

This disease has been prevalent throughout much of the same territory as peach yellows and has been especially serious in portions of Michigan, New Jersey and Pennsylvania.

Little peach is difficult to differentiate from yellows except in bearing trees, but in this stage may be recognized by: (1) *Fruit effects*, especially the undersize and the delayed maturity, varying from slight to 10 days or more. The flavor is inferior, sometimes watery and insipid, the flesh stringy in some varieties and the affected fruits are more or less flattened and somewhat rectangular. (2) *Vegetative effects.* The leaf abnormalities are very similar to those described for yellows, but the absence of willow or broom shoots will serve to differentiate little peach from

Fig. 196.—Normal and little-peach specimens of Greensboro. (*From N. J. Agr. Exp. Sta. Bul.* 356.)

yellows. Trees may sometimes be affected with both yellows and little peach, although mixed infections are not common.

The progress of the disease is variable, but there is a general decline in vigor and trees once affected never recover. Affected trees may continue to bear fruit, but it becomes smaller and poorer, the branches die back and the tree finally succumbs.

The disease can be transmitted from diseased to healthy trees by budding, as for peach yellows, but they are considered distinct and independent diseases. There is a deep-seated interference with the nutrition of the affected trees, which is visibly expressed by the recorded symptoms. No insect vector has been discovered. The immediate removal and destruction of the diseased trees is the only means of control.

ROSETTE

This virous disease of the peach is known chiefly from Georgia, South Carolina, Alabama, Missouri and Tennessee, but a few cases have been reported from Florida and some of the Mississippi valley states as far north as Illinois and Kansas. The only outside record is from Italy.

The characteristic symptom is the development of terminal or lateral tufts or rosettes, which may contain several hundred small leaves on

Fig. 197.—Twigs from peaches affected by rosette. (*After M. A. Blake, N. J. Agr. Exp. Sta. Bul.* **356**.)

axes not more than 2 inches long. These leaves are yellowish green or olivaceous, the outer ones with inrolled margins and a peculiar stiff appearance. The outer leaves turn yellow in early summer and drop as though it were autumn. A tree suffering from a general infection will always succumb during the following autumn or winter, but in partial infections the diseased limb dies, while the remainder develops the trouble the next season.

Rosette is infectious by either buds or grafts, but no positive insect vector has been discovered. The disease has been transferred experi-

mentally to apricots, wild and cultivated plums, cherry and sand cherry with the appearance of a mosaiclike mottle on apricots and plums. Marianna plum is listed as immune. Removal of the diseased trees is the only known control.

PHONY PEACH

This disease of peach is prevalent in the southeastern United States in the area extending from North Carolina, Tennessee, Missouri, also southern Illinois and eastern Texas to the other Gulf states, but has reached its greatest severity in the orchards of middle Georgia.

The principal symptoms and effects of the disease are: (1) a reduction in the size and number of fruits produced per tree; (2) dwarfing of the tree becoming more and more pronounced with increasing age; (3) a shortened terminal growth resulting in a more compact or congested head and denser foliage than normal, with leaves several shades deeper green; and (4) earlier flowering and foliage development than normal trees, and a tendency to hold the foliage some days longer in the fall. Infected trees do not die of the disease, neither do they recover, but they cease to produce fruit of quality and value and are a constant danger to uninfected trees unless removed. The onset of the disease in new plantings may prevent them from ever reaching commercial production, and bearing orchards in the most seriously affected areas may become 99 per cent phony in twelve to fifteen years after the first establishment of the disease.

The infectious character of the disease has been proved by grafting: normal scions on a phony tree become diseased; but phony scions develop normally on a tree free from the disease. The disease has not been communicated by patch grafting, which would support the belief that the virus is localized in the woody cylinder. Many tests have shown, however, that the disease may be easily communicated by root grafting "even minute portions of roots of diseased trees being sufficient to infect healthy trees." Following artificial inoculation, an incubation period of about 18 months is required before the symptoms of the disease become evident. There is no doubt as to the virous nature of the disease, and the existence of an insect vector, but positive identification has not been established, although the peach borer is suspected on the basis of circumstantial evidence. The disease has been artificially inoculated into plum, apricot and almond.

Neither resistant varieties nor resistant rootstocks have been found, and control can only be accomplished by the removal and destruction of diseased trees from the commercial orchard, as well as the destruction of abandoned orchards, neglected trees on home grounds or seedling

trees that have run wild in out-of-the-way-places. The virus is inactivated by immersion in water at 48°C. for 40 minutes or longer. The long period of incubation makes repeated inspections necessary in order that all infected trees may be eliminated. The disease cannot be spread by phony fruits, pits, buds or scions, as the infective principle is located only in the roots of the diseased trees.

RED SUTURE

This disease has recently become prominent in the peach belt of western Michigan and is reported to be well established in scattered localities. The disease is most apparent at ripening time, but the symptoms are not confined to the fruit. The following features may be recognized: (1) *Fruit characters.* Premature ripening (several days), first on the suture or crease side; a bumpy or rough contour, sometimes almost warty, with ridges parallel to the suture; and a deep red or purple color over the exposed side. (2) *Leaf changes.* The tips of some leaves and entire leaves of the smaller sizes are often lighter than normal, and the leaves in advanced cases show more downward curving and twisting of the petiole, with varietal variations. (3) *Terminal growth.* This is shorter than normal, the diameter smaller than normal, leaf clusters may sprout from most of the buds and an increased number of lateral shoots may develop along the main branches. This type of growth has been described as "feathering" or "fuzziness" and is somewhat similar to a condition found in little peach. In a well-developed case the entire tree appears somewhat yellowish or bronzed.

The infectious character of the disease has been proved by budding but rather limited tests have been made. No evidence is available as to an insect vector.

MOSAIC

The mosaic disease of peach was first reported from Texas and Colorado almost simultaneously but has since been recorded from Utah, California, New Mexico and Arizona. All fruit and ornamental varieties are affected and also the nectarine.

The following are the symptoms and effects: (1) *Color breaking in blossom petals.* There is an "abnormal breaking of the solid pink into islets, striations and broad streaks or splashes of pink color, definitely demarked by fainter pink or white lines," a character especially marked in varieties with large pink blossoms. Petals may also be crinkled or dwarfed. (2) *Retarded development of the foliage.* This may be confined to a single limb of a tree, or all but one limb of a tree may be involved, while others may show intermediate degrees of infection. There may

be some dwarfing of twig growth, a rosetting of leaf tufts and some dropping of leaves. (3) *Leaf mottling and deformation.* This effect will vary with the variety and the season. The mosaic symptoms may show as yellow and green patterns from tiny flecks to spots or blotches or even streaks, and in some varieties there may be a veinlet clearing (Elberta and J. H. Hale). Leaves may be reduced in size, narrow, crinkled, irregular in outline, owing to death of marginal portions, and develop some shot hole. The mottling becomes less pronounced as the season advances. (4) *Fruit abnormalities.* These include roughness or bumpiness marked by round or elongated bumps and depressions, reduced size, delayed ripening (week or more), inferior texture and flavor and an increased tendency to sunburn and cracking. (5) *Twig symptoms* are shorter internodes that may be stubby, thick or swollen.

The effect on fruit production results in fewer fruits, and these are reduced in size and of such low grade that many are not marketable.

The relation of mosaic or mosaiclike viroses on almond, apricot, plum and prune to typical peach mosaic is not entirely clear. It is worthy of note that plum and prune orchards adjacent to peach orchards showing 75 per cent mosaic have remained free from infection. Some cross inoculations have yielded variable results rather difficult to interpret, and there are some indications that there may be two or more strains of peach mosaic. In late studies (Bodine and Durrell, 1941) naturally occurring mosaics on other Prunus hosts were not transmitted to peach by grafting, but it is reported that scions taken from Maynard plum showing no virous symptoms when bud- or root-grafted on peach gave a typical mosaic. Scions from apricot, prune, plum or Myrobalan stock affected with a naturally occurring mosaic when grafted on J. H. Hale peaches gave a typical mosaic, and the same varieties of nursery trees when inoculated with buds from peach mosaic trees became symptomless carriers.

All attempts to transmit mosaic by juice inoculations have failed, and no definite vectors have been proved, although the rapidity of spread would indicate such a relation. Budding or grafting, however, give a very high percentage of infection. The heat relations of the various peach viroses indicate that mosaic is not closely related to the other peach viroses, since the former can be inactivated at 34.4 to 36.3°C., while mosaic is not inactivated by temperatures up to 50°C., close to the endurance of peach tissue.

Control.—Early spring inspections followed by the removal and destruction of infected trees must be practiced, followed by later inspections to detect other trees in which the symptom expression has been delayed. Cutting out of only the evidently infected branches does not give a satisfactory control.

OTHER PEACH VIROSES

Additional virous diseases of the peach have recently come into prominence. Mention may be made especially of the X disease or yellow-red virosis prevalent in the eastern peach-growing sections and the Western X disease from regions west of the Rocky Mountains. The former has been reported from Massachusetts, Connecticut, New York, Canada, New Jersey, Delaware, Maryland, Virginia and as far west as Michigan. Eleven other peach viroses have been recorded under specific names, but it is uncertain as to how many represent distinct virous entities (Hildebrand *et al.*, 1942). Although these troubles appear to be of increasing importance, our knowledge concerning them is too incomplete to justify any more detailed consideration.

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CURLY TOP

This virous disease of the beet and numerous other hosts has been referred to on beets at various times and places as the "California beet

disease," "western blight," "blight," "whiskered beets," "hairy root" and "curly leaf" but is now generally known as "curly top." The last name has seemed most appropriate, because rolling and curling of the leaves are the most striking effect of the disease.

The cause of this disease remained a mystery until about 1906 (Ball) when attention was first directed to the beet leaf hopper as causally related, and a few years later the relationship was proved. This finding was soon confirmed by other workers (1910-1917). The years following have yielded a continuous output of researches by Carsner and associates of the Federal Laboratory at Riverside, California, and other California workers. Since the discovery that the western blight or yellows of tomato was caused by the same virus (1927), the host range has been rapidly extended.

Curly top is primarily a disease of the arid or semiarid portions of North America west of the Rocky Mountains, its occurrence being limited by the range of its vector, the beet leaf hopper.

Symptoms and Effects.—The following are the recognized responses of the beet foliage to infection: (1) leaf curling; (2) blisterlike elevations on the leaves; (3) transparent venation of the innermost or youngest leaves; (4) wart- or knotlike swellings on the veins of the lower surface; (5) the exudation of a viscid sweetish liquid from the petiole, midrib or veins, clear at first but later dark and drying to form a brown crust; (6) yellowing and blighting of the leaves; and (7) a retardation of growth. The earliest symptom of the disease is an inward rolling of the lower and outer margin of the youngest leaves or sometimes an outward rolling of the margins or even a combination of the two types of curling.

The effects on the beet root are (1) the production of an increased number of lateral rootlets, giving the condition suggesting the common names of "hairy or woolly root" or "whiskered beets"; (2) a necrosis of the phloem extending throughout the vascular system and evident in the cross section of the root as dark concentric rings (Fig. 199); (3) a reduction in the size of the roots and a reduced sugar content but, in the moderate degrees of infection, not involving killing; and (4) shriveled, dead or rotted roots as an accompaniment of severe blighting and sun scorching of the foliage.

The effects on seed beets are (1) the production of "dead heads" or of blighted or dwarfed seed stalks and (2) reduced yields of seed of poor viability.

The degree of injury by curly top is influenced by the time of infection. Young seedlings may be killed outright; but if the infection is delayed, only the newly formed leaves will exhibit the symptoms described. Infections occurring late in the season may not be evident, but such beets if used for stechlings would develop only diseased seed stalks.

Curly top on garden vegetables, such as tomato, bean, squash, etc., often kills the plants in the seedling stage or in older plants causes the development of dwarfed and crippled, chlorotic individuals which may die prematurely. The response of tomato plants is very characteristic, including retarded growth, upward rolling of the leaflets, more or less chlorosis, a rigidity or harshness of the foliage, premature ripening of the fruit and frequently a death of the plant before killing frosts. Crop losses from curly top may vary from slight injury to complete failure.

Etiology.—Curly top is a virous disease which depends upon the beet leaf hopper (*Eutettix tenellus* Baker), for its dissemination under natural

Fig. 198.—Appearance of severe curly top of young sugar beet. (*After Eubanks Carsner.*)

field conditions. This relationship of the leaf hopper to the disease has been repeatedly demonstrated since the first work of Ball (1906, 1909). The severity of the disease in any environment will depend upon the abundance of the leaf hoppers and the time of their appearance. In nature only leaf hoppers that have fed on a wild or cultivated host harboring the disease become viruliferous, that is, are able to transmit the disease when they feed on healthy suscepts. Newly hatched nymphs cannot transmit the disease until they have fed on diseased foliage; and, in all cases, a period of incubation in the body of the hopper is necessary before infection will result. This "varies within quite wide limits, from the so-called 'normal incubation period,' or the period when at least 50 per cent of infection should be obtained, down to a single infection in as

short a time as 1 hour." Infection may be transmitted by a single hop-
per, but the action of numbers, or mass action, is very much more effec-
tive. The incubation within the host varies from seven to fourteen days,
with occasionally an earlier onset of symptoms (four days). Seedlings
are almost entirely invaded in two to four days, but in larger plants the
movement is slower.

FIG. 199.—Phloem necrosis in sugar beet affected with curly top.   (*After Carsner and Stahl,
Jour. Agr. Res.* **28.**)

The onset of the disease in any season may depend on the holding
over of a few viruliferous adults which feed on the newly planted crop
and start the infection, or the spring broods may obtain their virus from
infected wild plants in their natural breeding areas or even from cultivated
symptomless carriers.

The difficulty of artificial transmission was the main reason that the
virous nature of the disease was not recognized earlier. This applies to
the beet and also to the numerous other hosts. Since the nature of the
disease has been established, it has been possible to secure a low per-
centage of infection by juice inoculations. The causative principle is
generally distributed in both foliage and roots. In feeding, the mouth
parts of the hopper penetrate the phloem of the veins, and the introduced
virus travels largely along the phloem elements. The movement of the

virus is more rapid downward than up in tests with tobacco, and there appears to be a correlation between the virus movement and food translocation. This has been demonstrated by ringing experiments with tobacco plants. The movement is faster in beet than in tobacco. The infectiveness of the juice of a suscept is increased by an increase in the number of hoppers which have fed upon it, and, accordingly, the severity of onset of the disease is increased by a high population of leaf hoppers. The virus shows varying degrees of resistance to aging under different conditions: active for ten months in dried-phloem exudate, six months in dried leaf hoppers, four months in dried beet tissue, for only a few days in extracted beet juice exposed to the air, but for a much longer time under anaerobic conditions.

The disease has been transmitted by leaf hoppers which have fed on (1) a suspension of crushed viruliferous leaf hoppers; (2) diseased beet juice in 1 per cent aqueous solution of sucrose; and (3) a solution on which viruliferous hoppers had previously fed. The virus does not multiply in the leaf hoppers, and they act merely as internal mechanical carriers, although the virus is present in blood, salivary glands, alimentary tract and feces. Until recently, *Eutettix tenellus* was supposed to be the only vector, but this specificity has been denied by the discovery that another leaf hopper, *Agallia sticticollis*, is a common vector in the Argentine.

Host Relations.—The curly-top virus affects many species of plants, both wild and cultivated. The disease has been recorded in either mild or severe form as natural infections on the following cultivated species: garden and sugar beets, mangels, Swiss chard and spinach (Chenopodiaceae); common and Lima beans, cowpeas and alfalfa (Leguminosae); pumpkins, squash, watermelon, cucumber, muskmelon and cantaloupe (Cucurbitaceae); potato, tomato and pepper (Solanaceae); horse-radish, radish, cabbage and turnip (Brassicaceae); parsley (Umbelliferae) and 15 species of cultivated ornamentals including zinnia, African daisy, etc. It has also been transferred experimentally to 90 species of ornamental flowering plants.

In addition to the cultivated hosts, the disease has been reported on 14 species of wild hosts, and 57 species of weeds have been experimentally infected. Under California conditions, the virus has been shown to overwinter in 11 wild annuals, 3 perennials and also in alfalfa, parsley and potato. It is certain that some of these overwintering hosts are sources of the virus for the spring crops of leaf hoppers. Some of these important weed hosts are various species of Atriplex, certain species of Chenopodium, Russian thistle, amaranths, ground cherry, deadly nightshade, charlock, shepherd's-purse, knotweed and other species of Polygonum, dwarf mallow, cheese weed, alfilaria, or filaree and oxalis (*Oxalis stricta*). Some weed hosts under certain conditions (Russian thistle) may become symptomless carriers.

·Individual selections of sugar beets have shown varying susceptibility to curly top, and, in some cases, the virus has become so attenuated that beets carrying it do not show visible evidence of its presence. It has been shown that apparently healthy resistant beets may then act as symptomless carriers and yield the virus to leaf hoppers that feed upon them. Recent studies (Giddings, 1938) have shown the existence of at least four strains of virus by their reaction on different beet strains. The virulence has been attenuated by passing through a certain wild host and restored by passage through another, for example, through *Chenopodium murale* and then through sugar-beet seedlings at the cotylednary stage.

Resistance has been found in certain sugar-beet selections which have been propagated and introduced into the commercial fields. United States No. 1 selection produced an average of 18 tons in tests in four states as compared with 12 tons for commercial-run stock in the same fields. Later tests in three states showed an average of 13.5 tons for U. S. No. 1 and 5.47 tons from commercial stock. U. S. Nos. 33 and 34, selections from No. 1, gave a higher yield than No. 1 in more recent tests. Number A-600, a commercial strain, has shown resistance equal to or superior to U. S. 33 and 34. U. S. 12 is even better for resistance than U. S. 33 or 34 and shows less tendency to bolting under some conditions. The sucrose percentage in U. S. 12 is equal or superior to U. S. 34 or A-600, but inferior to U. S. 33. U. S. 12 and U. S. 33 hold a dominant position pending further improved varieties. The availability of these resistant selections is causing the return of sugar-beet culture to ·some sections in which this crop had been discontinued because of the severity of curly top.

No varieties of tomato have shown any appreciable degree of resistance, and losses in this crop are frequently heavy, in the most severe incidence amounting to 100 per cent infection before the end of the season. Reduction of losses has been accomplished by (1) shading by use of slat, or muslin covers, an interplanted tall crop or only temporary cloth shade; (2) delayed setting in the field; (3) planting the seed directly in the field to avoid transplanting; and (4) keeping tomatoes well separated from beets. ·Marked resistance has been found among beans, squash and pumpkins, represented by the Burtner Blightless, California Pink, California Red Jenkins and Red Mexican among field beans, the Yakima and Umatilla Marblehead squashes, and Sweet Cheese and some other varieties of pumpkin (*Cucurbita maxima*).

Control.—The high temperature, bright sunshine and low humidity of the semiarid regions of America west of the Rocky Mountains offer conditions for the development of large numbers of insect vectors and the expression of diseased conditions. The severity of the disease on the

various hosts fluctuates with the abundance and virulence of the beet leaf hoppers, and consequently seasonal variations in severity are experienced. Little can be done in modifying the environment or reducing the numbers of leaf hoppers by either artificial or biological methods. A special study has been made of practices affecting the breeding areas in southern Idaho with attention to (1) elimination of intermittent farming and the subsequent use for grazing of lands so released; (2) the prevention of grazing on the worst of the weedy lands; (3) the restriction of grazing on the less weedy lands; and (4) attention to miscellaneous practices including the prevention of fires, the preservation of the sagebrush or other desert shrubs rather than using them for firewood, and the control of rodents. Some relief has been obtained by cultural practices, such as early planting (sugar beet and tomato), providing a shade crop (tomato) or following other selected cultural practices (see previous paragraph). The development of the resistant strains of sugar beet has given new life to the industry, and it is hoped that resistance will be maintained.

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440 VIROUS DISEASES

WALLACE, J. M., and MURPHY, A. M.   U. S. Dept. Agr. Tech. Bul. 624: 1–47.   1938.
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ASTER YELLOWS

The yellows of asters (*Callistephus chinensis* Nees) was widespread and destructive in the eastern United States as early as 1902 but has come into more prominence in recent years as a handicap to the cultivation of asters in certain sections.   It is now prevalent in many areas throughout North America, its occurrence being limited only by the absence of the insect vector.   The disease has been reported from Bermuda, Hungary, Germany and Japan and undoubtedly occurs in other countries.

Symptoms and Effects.—The disease is systemic and produces profound changes from the normal as follows: (1) *Chlorosis.*   The first symptom of the onset of the disease is a slight yellowing along the veins, or a vein clearing, in the whole or a part of a single young leaf.   This is followed by the same symptom on the new leaves as they develop, until, later, the new leaves are chlorotic throughout.   This chlorosis may be sectorial, that is, the leaf tissue on one side of the midrib or the leaves on one side of the plant may be chlorotic before the remaining portions are affected.   Leaves mature at the time of infection remain green.   Necrosis of portions of the yellowed leaves or of the stem tissues a short distance below the apical buds may occur in advanced stages of the disease.   (2) *Dwarfing.*   The degree of dwarfing varies with the age at the time of infection.   It is very pronounced in young plants, resulting in a bushy growth with shortened internodes and, in extreme cases, a failure to flower.   (3) *Upright habit.*   Leaves and individual branches are more erect than is normal for the variety, and there is an abnormal production of secondary shoots which also are more erect than is normal for the variety.   (4) *Malformation.*   Leaf blades may be smaller and narrower and the petioles longer than normal; flower changes include a general greening of parts, transformation of ray florets to the tubular type, sterility and malformation of disk florets and general tendency for flower parts, the pappus and the bracts to become leaflike.

Affected plants are not killed, but they are so seriously crippled that they are practically worthless for flower production.   Only a few plants may contract the disease, or in the very severe cases even 90 to 95 per cent may be affected, while 100 per cent infection has been observed in extensive plantings.

Etiology.—Aster yellows is an infectious virous disease that is introduced into growing plants by the feeding punctures of the leaf hopper, *Cicadula divisa* Uhl. (*Macrosteles divisus*).   This vector was at first incorrectly assigned to the European species, *C. sexnotata* Fall.   The

similarity of aster yellows to certain insect-borne virous diseases of other plants pointed to an insect relation, and this was proved by Kunkel in 1926. The disease is transmitted by budding but not by other mechanical means and is not seed-borne. Another leaf hopper, *Thamnotettix montanus*, has been shown to transmit yellows of aster to a limited extent in California, but *T. geminatus*, which transfers the disease to celery and carrots, will not carry it to asters.

The leaf hoppers overwinter in the egg stage, and freshly hatched hoppers are free from the virus while young seedlings are free from the yellows until they have been fed upon by viruliferous hoppers. Nonviruliferous hoppers become carriers by feeding on certain infected biennial or perennial host plants which carry the virus over the winter. Overwintering hosts are found in the open and also in greenhouse cultures. The disease is transmitted by the feeding punctures of both nymphs and adults, but an incubation period of about ten days is required before they become inoculative. For the disease in the aster plants, the incubation period before the development of visible symptoms is variable, and on the basis of numerous tests thirty-nine days is the maximum and about eighteen days the average. After feeding upon an infected plant, many of the hoppers are able to transmit the disease as long as they live, while some seem to lose their infective power. The virus has been transferred mechanically from viruliferous hoppers to nonviruliferous hoppers which were then able to transmit the disease as effectively as those fed on infected plants. Evidence has been presented which has led to the conclusion that the virus actually multiplies or increases in amount within the vector (Black, 1939, 1941).

The severity of the disease upon the China aster may be explained by the fact that it is a favorite host of the leaf hopper and that the aster is very susceptible. Sufficiently early in the life of the asters, a good supply of viruliferous hoppers have become available unless their numbers are reduced by unfavorable weather conditions or diseases and predaceous enemies.

The disease spreads more during the last part of the season owing to the inactivation of the virus during the high summer temperatures. If the vectors are exposed to temperatures of 31 to 32°C. up to eleven days, they temporarily lose their ability to transmit the disease, but after twelve or more days exposure they are permanently unable to transmit the disease. At least 12 mild strains have been isolated by heat treatment and these are characterized by less severe chlorosis, slight erectness of growth, little stunting or distortion and less branching of secondary shoots (Kunkel, 1937).

Host Relations.—Since the discovery of the insect vector, aster yellows has been transmitted experimentally to a large number of other

suscepts. In the first detailed study, Kunkel recorded over 50 different species in 23 different families of plants and, in a later study, reported experimental transfer to 120 more species in 30 different families, but there is no record of the natural occurrence of yellows on most of the species experimentally infected. The number has been still further increased by the studies of Severin in California. Practically all of the reported suscepts are dicotyledonous plants, and about one-half of the total number belong to the Compositae, many being cultivated ornamentals. "Immunity, resistance and a high degree of susceptibility are in some instances shown by closely related species," and there is great diversity of symptoms and effects.

Information as to the natural occurrence on overwintering hosts is very incomplete. It is certain that the virus lives overwinter in some of its wild hosts, including wild carrots, sow thistles, fall dandelions, daisies, and chrysanthemums and others in the eastern United States.

The most important crop plants seriously affected with yellows are carrots, celery and lettuce, the disease on the latter host having been known in New York as white heart or rabbit-ear and in Texas as the Rio Grande disease.

Some differences of performance have been shown by the virus from asters by different workers. Kunkel in New York was unable to infect carrots, celery, potatoes and zinnias by means of the insect vector, but Severin has reported successful infections on these hosts in California. There has been some doubt, therefore, as to whether a single virus entity was responsible for these different responses. On the basis of the performance of other viruses, it would seem that the California yellows is only a strain of aster yellows rather than a distinct entity.

Control.—It has been pointed out that the disease is less severe in plots surrounded by cultivated fields, than in those adjacent to pastures or wastelands, because of a smaller population of the vectors. Several different methods for the prevention or at least reduction of yellows in asters have been suggested: (1) the elimination of overwintering hosts from the vicinity of aster plantings; (2) the removal and destruction of aster plants as soon as symptoms of the disease are apparent; (3) the spraying or dusting of the plants for the destruction or repulsion of the leaf hoppers; and (4) the partial or complete exclusion of the leaf hoppers by some sort of barrier. The first three measures may afford limited protection but with a heavy population of hoppers are of but little value.

A number of different methods of exclusion of the leaf hoppers have been used: (1) A wire-screen fence, 18 meshes to the inch, and at least 5 or 6 feet high completely surrounding the planting or a similar fence of tobacco shade cloth. An enclosure of this type, supplemented by rogu-

ing, has held losses to 20 per cent as contrasted to 80 per cent in unprotected controls in New York, but it has not been quite so successful in Wisconsin. Small enclosures have given more protection than large ones. (2) A complete enclosure or an aster tent supported on posts 6 feet high, using a screening cloth not coarser than 22 by 22 threads per inch. With care to make these tents completely insectproof and care in entering them, they have afforded perfect protection, and have been used with success in experimental work and by commercial growers.

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POTATO VIROSES

It seems probable that virous diseases of the potato have been prevalent ever since the so-called running out or degeneration of varieties has been recognized. Mosaics undoubtedly formed a part of the symptomatic complex included in the *Kräuselkrankheiten* or "curl" of the Germans, which undoubtedly included also leaf roll, later recognized as a separate and distinct disease. There was a severe development of leaf roll in Germany in 1907, and attention to leaf roll and mosaic in America soon followed as a result of the observations of Orton, of the U. S. Department of Agriculture, in Europe and later extensive travel through the important potato sections of the United States. His special publication on "potato wilt, leaf roll and related diseases" appeared in 1914 and gave abundant evidence that leaf roll and mosaic were prevalent and rather widespread in the important potato districts of the United States from Maine to Colorado. Since this time, workers in various countries have given more and more attention to virous diseases of the potato until at present there is a voluminous and in some cases bewildering literature on the subject, involving over 300 separate articles which have appeared since 1914. In addition to leaf roll and mosaic and its complexes, various other virous diseases of the potato of lesser importance have been recognized.

LEAF ROLL

On the basis of the numerous reports, it seems probable that leaf roll is coexistent with the cultivation of the potato but that it reaches its greatest severity only in regions in which its insect carriers are especially abundant.   These carriers appear to decrease in the northern latitudes or higher altitudes, which would explain the greater freedom of cooler sections from leaf roll.

Symptoms and Effects.—The following are characteristic features of the disease: (1) *Rolling of the leaves.*   The leaflets curl upward from the margin toward the midrib and in extreme cases become nearly tubular,

Fig. 200.—Potato plant showing a well-developed case of leaf roll.   (*Photograph by B. F. Dana.*)

and exhibit a rigid character or have a hard or crisp "feel."   When the disease is contracted during the growing season, the rolling of leaflets may be confined to the upper parts of the plant (primary leaf roll), and the petioles of the affected leaves are frequently raised to form a more acute angle with the stalks than in normal plants.   When infected tubers are planted, the lower leaves invariably show the first rolling of the leaflets (secondary leaf roll), and the trouble advances until the entire plant may show the symptom.   (2) *Change of foliage color.*   Early stages of leaf roll may show but a slight pallor, while later in the season the color may be of a pronounced yellowish cast, and in many cases the affected leaves will show also reddish or purplish tints.   These expressions vary with the variety and are modified by environmental conditions and the

severity of the infection. (3) *Change of form.* Affected plants may be low-spreading or compressed in form or in some cases more slender with an abnormally erect V-shaped form, with general reduction in size and length of life. (4) *Endurance of the seed piece.* This feature is common

FIG. 201.—Single potato leaf showing the characteristic rolling of the leaflets from a plant affected with leaf roll. (*Photograph by B. F. Dana.*)

in leaf roll, but is not a diagnostic character. (5 (*Reduction in size of tubers.* This is an invariable feature, and the tubers are borne in clusters close to the stem or directly upon it owing to the shortening of the stolons. (6) *Phloem necrosis.* Necrosis of the phloem in the stems of leaf-roll plants is of universal occurrence, and with severe external symptoms the dead elements may pervade the entire plant. *Net necrosis* of tubers is a

phloem necrosis which frequently develops in dormant tubers and has been shown to be an accompaniment of leaf roll. Stem-end browning involves both phloem and xylem, is generally darker in color and does not extend as deep. *Spindling sprouts* from leaf-roll tubers are not uncommon. Neither phloem necrosis nor spindling sprout are diagnostic

Fig. 202.—Spindling sprout, a symptom which frequently accompanies leaf roll. (*Photograph by B. F. Dana.*)

characters, since they are not always in evidence and may be caused by entirely different factors (unfavorable temperatures, etc.). (7) *Starch accumulation.* In leaf-roll plants starch (and also glucose) accumulates in the leaves, since it is slowly if at all carried to the tubers, while normal leaves are free from starch in the morning due to translocation during the night period.

Leaf roll is responsible for very pronounced reductions in total yields and further losses from small or irregular tubers that are not marketable.

Estimates by authorities in this and foreign countries give yield reductions ranging from 20 to 90 per cent, even without the added effect of other potato viroses. Varieties have been grouped as: (1) highly susceptible; (2) intermediate in susceptibility; and (3) least affected. In a recent study (Loughnane, 1941) Flourball, Arran Banner and Majestic were classed in the last group. In comparative tests (Tuthill and Decker, 1941) Cobbler was more resistant (23 per cent leaf roll) than Chippewa (35 per cent).

Etiology.—Leaf roll is now recognized as a definite and specific virous disease. Space will permit only a few of the salient features in the devel-

FIG. 203.—Cross and longitudinal sections of potato tubers showing net type of phloem necrosis.

opment of our knowledge of the virous relations. The first important step after recognizing the disease as of the virous type was the demonstration of its transmission by grafting, and two different methods have been employed: (1) stalk grafting and (2) tuber grafting. The next important step was the demonstration of leaf-roll transmission by the feeding punctures of aphids (Botjes in Holland in 1920 and Schultz and Folsom in the United States in 1921). Field observations by various workers have repeatedly confirmed the importance of aphids in the natural dissemination and transmission of the disease, and these observations have shown that severity and spread of the disease is in accordance with abundance of the insect vectors. Maximum infection can be expected if the temperature and rainfall in June is normal while a hot and dry June may result in a moderate development of the disease.

The aphid, *Myzus persicae*, is the most efficient vector, but a number of other species (*M. circumflexus, Aphis rhamni, A. fabae* and possibly

others) are considered as poorer vectors. It has been shown definitely that the incidence of infection is increased with an increase in the population of aphids but that the disease can be contracted with only a small number of viruliferous aphids per plant (2 to 6). The development of the disease in the field may be from seed tubers already carrying the virus or from new infections from the feeding punctures of some of the vectors. The disease cannot be transmitted like the mosaics by leaf mutilation or by juice inoculations.

If viruliferous aphids feed upon potato plants too late in the season, the disease may be transmitted but without any appearance of symptoms until the tubers are used for seed next year. The performance of varieties in this respect varies as may be illustrated by one case (Aaran Comrade) in which current-season symptoms did not appear if the infection was more than eight weeks after planting, while in another variety (Kerr's Pink), infections more than five weeks after planting remained latent until next season. In current-season, or primary, infections, not all of the tubers will be carriers, especially in late infections, due apparently to the slow downward movement of the virus.

Two minor forms of leaf roll have been recognized: (1) *marginal leaf roll* of Quanjer (1923) which may not be a distinct virosis; and (2) *apical leaf roll* of Schultz and Bonde (1929) characterized by slight dwarfing, rolling of the upper leaves as in primary infections, but persisting in this form in succeeding generations.

MOSAICS AND MOSAIC COMPLEXES

When potato mosaic was first recognized as a virous disease distinct from leaf roll, the opinion prevailed that it represented a single disease entity. As investigations were continued, a great diversity of symptomatic expressions began to accumulate and the literature of recent times is filled with a confusing medley of names, some of which represent distinct or specific viruses while others are simply variations of a single virous entity modified by host or other influences, or of the combined effect of two or sometimes even more virous entities. Mosaics and mosaic complexes are now prevalent to a greater or less extent throughout the world.

Symptoms and Effects.—The following are characteristic for the potato mosaics or mosaic complexes on the potato, but it should be pointed out that still other responses may be induced by some of these viruses on other hosts: (1) *Mottling*, a localized chlorosis or spotting of the leaf blades by light-green areas which vary in shade, form and size. The spots may vary from a very slight fading of the green to almost a pure yellow and may be punctate, elongate, circular, angular or irregular. They seldom exceed a few millimeters in any dimension and, when faint,

are most evident when shaded. The chlorotic areas are thinner than
the surrounding green tissue, the palisade parenchyma consisting of very
short or almost cubical cells as contrasted with the much elongated pali-
sade cells of a normal leaf. (2) *Veinbanding.* Slightly pale areas or
patches develop chiefly between the veins, while the tissues directly in
contact with the veins remain green. (3) *Wrinkling or rugosity of leaves.*
This is an abnormal unevenness of surface due to depressions and promi-
nences, in the former of irregular height and depth, in the latter of uni-
form height, but with the depressions only at the veins (see Fig. 204).
This unevenness of surface may be very slight, quite evident or extreme

Fig. 204.—Diagrammatic cross sections of tobacco leaves, showing effect of mosaic. *A*,
section through hypoplastic area of tobacco leaf; *B*, section through dark-green area,
showing hypertrophy of palisade tissue; *C*, section through ordinary dark-green tissue.
(*After B. T. Dickson, Macdonald Col. Tech. Bul.* **2.**)

to compare favorably with the normal foliage of Scotch kale or Savoy
cabbage. (4) *Ruffling.* An abnormal unevenness of the leaf blade,
caused by ridges that become more pronounced with passage from the
midrib to the lateral margins resulting in waviness of margin. Ruffling
may be slight or very pronounced. (5) *Curling.* This is an abnormal
bending of the leaf blade downward along the main vein. (6) *Leaf
rolling.* This may be similar to the effect described for true leaf roll
(upward rolling) and may occur with mottling and other accompanying
symptoms, or there may be a downward rolling. (7) *Stem and leaf
necrosis.* Three types of necrosis are recognized: *basal,* involving a group
of leaves at the base of the stem giving the effect described as leaf-drop
streak; *top necrosis,* or a killing of some of the terminal leaves; and *foliar
necrosis* or necrotic spotting and killing of leaves occupying an inter-
mediate position. There is an accompanying internal necrosis in petioles
and stems. Necrosis may appear on the undersides of the veins, increase
in severity and spread along the veins causing brown blotches on the

leaves and streaks on the petioles and stems. Later the entire leaf may become dead and brown and collapse or drop down and remain hanging by the shriveled petiole (very characteristic of the basal type), or sometimes it may be detached by the wind. In other cases isolated necrotic spots on the leaves may be the first evidence of the disease, and may be followed by more complete necrosis as the disease advances. (8) *Tuber necrosis.* The appearance of internal brown dead spots involving either parenchyma or phloem or both. (9) *Cracking* or *splitting* and deforming of tubers. (10) *Dwarfing and early maturity.* The reduction in size of tops may vary from slight to extreme as illustrated by the type known as curly dwarf, while there is more or less reduction in size of the tubers and in some cases an impairment of quality. In affected plants the process of photosynthesis does not function so efficiently as in healthy plants, and as a result yields are reduced ranging from 16 to 25 per cent to as high as 65 to 85 per cent depending on virous entities involved.

Etiology.—Mosaics in their various forms are all infectious or communicable and tuber-perpetuated. The various mosaics or mosaic complexes differ somewhat as to their degrees of infectiousness, but they can all be transmitted from diseased to healthy plants. This transmission may be demonstrated by artificial means and takes place under natural growing conditions in the field. Transmission is successful by stem grafts, tuber grafts, juice inoculations and by the feeding punctures of aphids which have been pastured upon diseased plants.

A number of simple virous entities and virous complexes have been recognized, but a detailed consideration of the entire number is beyond the scope of this treatment. The following are of fundamental importance especially under American conditions:

1. *Mild or Common Mosaic.*—In this type the mottling may be very faint (supermild), mild or very distinct, with some leaf ruffling, slight dwarfing, and reduction in tuber size. It has been shown that in some cases potatoes showing mild mosaic contain two components in addition to the latent or X virus.

2. *X Types* (Includes "Mottle" of Johnson, or Latent).—This virus may be present in certain varieties of potatoes (apparently healthy or virus-free) without a visible expression of symptoms. Its presence has been demonstrated in all commercial American stock by juice inoculations from apparently virus-free stock to healthy tobacco and tomato, causing a faint mottle or at other times fairly distinct interveinal chlorotic areas. Necrotic symptoms may be produced in certain cases by successive transfers from healthy stock, or the latent of some varieties will produce mottling and necrotic symptoms of varying severity when transferred to other varieties. In England some varieties carry the virus without

Fɪɢ. 205.—*A*, an apparently healthy leaf of Early Rose affected with latent virus; *B*, leaf of Early Rose affected with rugose mosaic; *C*, healthy leaf from potato seedling; *D*, leaf from potato-seedling plant affected with veinbanding virus. (*After Burnett and Jones, Wash. Agr. Exp. Sta. Bul.* **259**.)

any, visible expression, while other varieties develop mild mottle, and others severe mottle and necrosis. This virus is transmitted by core grafts and by juice inoculation, but it is not insect-transmitted. Experimental infections have been produced on tomato, tobacco, pepper, Jimson weed, henbane, etc. (virus *X* of Smith apparently includes the common mosaic). At least six strains of virus *X* have been differentiated

FIG. 206.—Severe form of rugose mosaic on Bliss Triumph. (*Photograph by B. F. Dana.*)

on the basis of reaction on test plants, different temperature inactivation points (68 or 75°C.) and different dilution end points.

3. *Y Types* (Includes Streak of Orton, Veinbanding of Valleau and Johnson, and Some Other Names).—On certain varieties this virus may produce only a mild mosaic mottle, others may carry it without symptoms, while in others it produces a typical "leaf-drop streak" effect [see (7) under Symptoms]. Four different strains of the *Y* type have been recognized (Köhler, 1940). *Y* types are sap-transmissible and are transmitted by *Myzus persicae* but apparently not by other aphids.

In addition to X and Y, a Z virus has also been recognized, while A, B, C, D and E occupy the other end of the alphabet. It is not recorded whether potatoes affected with these viruses have been used in the preparation of "alphabet soup."

4. *Potato Virous Complexes.*—The composite nature of certain potato virous diseases has been demonstrated by isolating the viruses which cause them and by inducing the disease by their combined action. *Rugose mosaic* is caused by the combined action of the X virus (latent) and the Y virus (veinbanding). This type is characterized by "distinct dwarfing, more chlorosis, and more diffused mottling, a more rugose type of wrinkling and a tendency to show brittleness, spotting, streaking, leaf dropping and premature death," with a marked reduction in tuber size. *Crinkle* studied by Smith in England is reported to contain X and Y, with evidence of a third constituent, which Murphy has called "virus A." Since all American seed stock contains virus X, the transmission of virus Y to this stock by the aphid vector will produce rugose mosaic. In composite viruses a varying expression of the complex is likely to occur as a result of environmental factors, variety peculiarities and varying virulence of the contributing viruses.

A frequently observed feature of the mosaics of potato is the masking of the symptoms as a result of exposure to certain environmental conditions. High temperatures, low air and soil moisture and intense sunshine, which usually occur together in many environments, may combine to cause almost a complete masking of symptoms. Short exposures to temperatures above the critical (23 to 24°C.) are sufficient to mask mosaic symptoms, the rate of masking depending upon the duration of the high temperature, the actual temperature prevailing and the age of the plants. Exposure to high temperatures does not destroy the infectious properties of the mosaic viruses, since aphid transmission has been shown to continue during the period of masking, and the progeny from masked cases will again exhibit the disease when grown under suitable conditions.

Aphids are the most important vectors of the potato mosaics, and the following have been tried and found guilty: *Myzus persicae, M. pseudosolani, Macrosiphum gei (-solanifolii), Aphis rhamni* and *A. fabae (rumicis)*. Some transfer has been reported by cabbage caterpillars and by the flea beetle (*Psyllioides affinis*).

OTHER POTATO VIROSES

In addition to leaf roll and the mosaics and mosaic complexes, the following specific virous diseases of the potato have been recognized:

Spindle Tuber.—This disease is characterized by: (1) a smaller, spindly, upright growth, often of a darker green or greyish color, with

leaves more erect than normal and sometimes narrower and showing some rugosity; and (2) tubers of an abnormal spindle shape, with conspicuous and numerous eyes, especially in elongated varieties like Netted Gem.   In market-size stock there is a decrease in the cross-section area relative to the length. , This disease is believed to be an important cause of the running out of potato stock in America in which poor tuber shape is a common character.   It has recently been reported from New South Wales.   "Giant hill" has been described as a phase of spindle tuber but some authorities doubt its virous origin and suggest the possibility of a genetic abnormality.

Unmottled Curly Dwarf.—This disease was at first believed to be a phase of spindle tuber, but opinions seem to be somewhat divided.   The symptoms are very similar, but certain differential characters have been claimed: internal necrosis of the tubers in the current season; absence of tuber elongation; and a downward curving of the leaf along the midrib, and rigidity and brittleness of stems and petioles, all features absent in spindle tuber.

Witches'-broom.—Some of the more outstanding characters are: (1) the production of an increased number of slender and in the extreme very dwarfed shoots of more upright habit bearing thin, light-green leaves of reduced size (various gradations down to simple); and (2) a marked reduction in size of tubers and their increase in numbers, with as many as 200 of varying size from peas to walnuts as the product of a single hill.   This disease has been described in Scotland as "wilding" or "semiwilding."

Psyllid Yellows.—The symptoms have been described as follows under conditions of unmodified light: "Yellowing, basal leaf rolling and purpling of younger leaves, yellowing and rolling of older leaves, nodal enlargement, increased axillary angle, aerial tubers and shoots, frequent rosetting, various apical growths, and distortion, excess tuberization and inhibition of the rest period."   (Richards and Blood, 1933.)   Tuber transmission is not obtained, and the full expression of the disease results only when the nymphs of the psyllid, *Paratrioza cockerelli* are allowed to feed continuously on the tissue of the infested plant.   Doubt has, therefore, been expressed as to the virous nature of the trouble, with the evidence indicating that a toxic substance is produced during the feeding process.

Calico.—Large, irregular blotches of various shades of yellow, generally rather bright, on the leaflets characterize this disease.   Plants are of normal size or only slightly reduced.   The disease is tuber-perpetuating and is transmissible by tuber grafting and by inoculation of leaves with unfiltered juice, but not with filtered juice.   The potato aphid (*Macrosiphum solanifolii*) is the vector.   The virus has been mechanically

transmitted to various other Solanaceae and to several legumes including beans, cowpeas, crimson, red and white clover and to cucumber. It is suggested that potato calico and alfalfa mosaic are strains of a single virus entity. The trouble has been much reduced by late planting.

Aucuba Mosaic.—A disease somewhat similar to calico showing the following symptoms: (1) small, round yellow spots which sometimes coalesce on the leaves; (2) net necrosis of the tubers; and (3) a cortex and pith necrosis in some varieties. Transmissible by tuber grafting and by leaf mutilation but no known insect vectors. The name "aucuba mosaic" has been applied because of variegations similar to the leaves of *Aucuba japonica*. Calico and aucuba mosaic are probably related. Canada streak and aucuba are either closely related or identical.

Yellow Top.—Characterized by distinct dwarfing, spindling shoots, and stiff leaf texture and sometimes by extreme chlorosis, tuber net necrosis and distinct rolling of leaves. Tuber-perpetuated and transmissible by grafts.

Yellow Dwarf.—The following are notable symptoms and effects: (1) some vein clearing and a mild mottle: (2) spindling and dwarfing of all parts; (3) water-soaked zones on the petals, necrosis of petals and sepals and failure of flowers to open; (4) top killed before blossoming or blossoms rare in the most acute stage; and (5) the nonemergence of plants from infected tubers.

The disease is transmitted by the clover leaf hopper (*Aceratagallia sanguinolenta*). Various species of Trifolium are susceptible, and there is some evidence that common red clover is an important source. The disease has been transferred by grafting or by the vector to various species of Nicotiana, to China aster, crimson clover, broad bean, eggplant and *Physalis pubescens*. Many different weeds (15) have been artificially infected, and the virus has been identified from natural infections in red clover, *Rudbeckia hirta*, *Barbarea vulgaris* and *Chrysanthemum leucanthemum* var. *pinnatifidum*. It is suggested that the latter is an even more important host than red clover. This is confirmed by the observations in some areas that there is no correlation between yellow dwarf epidemics and the abundance of red clover plantings.

The symptoms develop most rapidly at high temperatures but at 16°C. may be completely suppressed. The "poor-stand" phase is thought to be due to high soil temperatures and late planting. Seven different strains were studied by Black (1940) each of which produced different symptom expressions on *Nicotiana rustica*, but on Green Mountain potato all caused severe disease but of varying intensity. The incidence of the disease in 1935 varied from 1.6 per cent in U. S. D. A. No. 44537 to 48.4 per cent in Columbia Russet, but in 1936 the percentage dropped to only 2.4 in the latter.

Control practices suggested include: the isolation of seed plots from diseased clover and potatoes; test seed potatoes if, from a field which shows infections at the edges, or select seed from the middle of the field only and harvest early; use of disease-escaping or resistant varieties; and dusting or spraying for the control of the vector.

Curly Top.—The affected plants are stunted, with yellowish inward-rolled leaflets, and sometimes a downward curving of the petioles. Advanced stages of infection may show dwarfed axillary shoots, and final yellowing and death. This is caused by the virus of curly top of beet and other hosts and has been reported from natural field infections, limited to natural range of the beet leaf hopper (see beet curly top); also produced by artificial inoculations.

Control of Potato Viroses.—It should be emphasized first that potato seed stock which is free from viruses will probably remain free unless the viruses are introduced by the feeding of some of the insect carriers. Theoretically the eradication of insect carriers from our fields would be the first suggestion, but this presents such difficulties that it is not economically practical, since frequent applications of insecticides would be necessary throughout the entire growing period. The following are control measures which have given some success, although at times the results have been somewhat disappointing:

1. The use of certified seed or the regional selection of seed. Certified seed is stock that is grown under supervision of a state inspection system to ensure its freedom from virous and other diseases or of their presence in a certain allowable minimum. It is known that some commercial potato areas are more free of the various insect vectors than others and consequently may produce stock that will show a low percentage of infected seed. This is reported to apply to some of the more northern seed-potato sections of Michigan, Wisconsin, Minnesota and Montana.

2. The use of seed plots or special plantings which are systematically rogued throughout the season so as to hold the diseases down to a minimum. Such seed plots should be at least 500 feet from other potato fields, should not have grown a crop of potatoes for several years and should be kept free from weeds. Seed plots may be started from certified seed or still better from seed obtained by one or the other of two methods: (a) *Tuber indexing.* In this method one eye from each tuber is tested in the greenhouse hotbed, cold frame or in some southern localities in the open field early in the spring and all tubers rejected which are shown to be diseased by the performance of these single-eye plants. (b) *Hill indexing.* Select apparently healthy hills late in the growing season, and mark for use. At digging time select again and discard entire hill for "off form" or any evidences of virous diseases, and preserve in

numbered hill lots. These selections may be handled in two ways: (*a*) by planting in hill units, and discarding any units which develop virous symptoms, or (*b*) by testing one tuber from each hill in advance of the field planting. Varying degrees of success have followed from hill indexing.

3. The selection and breeding of resistant or immune varieties has occupied the attention of workers, probably less for leaf roll than for the mosaic and mosaic complexes. Differences in varietal susceptibility have been reported for leaf roll but no immune varieties have been found. It seems probable that some of these cases of apparent resistance are due to early maturity, and thus escaping infection, rather than to real resistance. This is the apparent explanation of reports that Early Ohio and Irish Cobbler are more resistant than the late Rural varieties. Under English conditions, Great Scot is very seldom infected.

The large number of mosaic viruses and complexes has made this aspect of control more difficult, for certain varieties may carry one virus without symptoms and be moderately or highly susceptible to others. The Irish Cobbler and Spaulding No. 4 have a reputation of resistance to mild mosaic, and Great Scot (in Ireland) has been reported resistant to mosaics as well as leaf roll. More recently Katahdin, Chippewa, Houma, Sebago, and Earlaine have been developed and show resistance to mild mosaic. Katahdin, a short elliptical to roundish tuber, although resistant to common mosaic, is susceptible to leaf roll and spindle tuber. This is valuable for commercial production in certain areas and as parent stock for breeding of other varieties.

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IMPORTANT DISEASES DUE TO VIRUSES

For key references on these diseases see F. D. Heald, "Manual of Plant Diseases," 2d ed., pp. 314–322. 1932.

Abaca or Manila hemp—Bunchy top
Abutilon—Infectious chlorosis
Alfalfa—Dwarf
Alfalfa—Mosaic
Alfalfa—Witches'-broom
Alfalfa—Yellow top
Amaryllis—Mosaic (*see* Hippeastrum)
Anemone—Alloiophylly
Anthurium—Mosaic
Apple—Bitter pit(?)
Apple—Infectious variegation
Apple—Mosaic
Artichoke, Globe(?)
Ash—Mosaic
Aster—Mosaic
Aster—Yellows
Avocado—Sun blotch
Banana—Bunchy top
Banana—Infectious chlorosis
Bean—Curly top
Bean—Mosaics
Bean, Adzuki—Mosaic
Beet—Curl disease
Beet—Curly top
Beet—Mosaic
Beet—Yellowing disease
Begonia—Mosaic
Black locust—Brooming disease
Blackberry—Dwarf
Blackberry—Mosaic
Burning bush—Infectious chlorosis
Cabbage—Mosaic
Cacao—Roncet
Cactaceae—Mosaics
Calendula—Rosette (peanut)
Calliopsis—Rosette (peanut)
Campanula—Spotted wilt (tomato)
Canna—Mosaic
Cantaloupe—(*see* Melon)
Cardamine—Mosaic
Carnation—Mosaic
Carrot—Yellows
Cassava—Mosaic or leprosy
Cauliflower—Mosaic
Celery—Mosaic
Celery—Yellows (*see* Aster)
Cherry—Buckskin

Cherry—Crinkle
Chicory—Mosaic
Chinese cabbage—Mosaic
Chrysanthemum—Yellows
Cineraria—Spotted Wilt (tomato)
Citrus—Brown spot (oranges)
Citrus—Crinkly leaf
Citrus—Decorticosis
Citrus—Exanthema
Citrus—Leprosis
Citrus—Peteca (lemons)
Citrus—Ring blotch or zonate chlorosis
Citrus—Wither tip(?)
Clover—Mosaics
Clover—Yellows
Coffee—Phloem necrosis
Commelina—Mosaic
Coreopsis—Yellows (aster)
Corn—Mosaic or stripe
Corn—Streak or variegation
Corn—Sugar-cane mosaic
Cosmos—Yellows (aster)
Cotton—Acromania or crazy top
Cotton—Leaf curl
Cotton—Stenosis or smalling
Cowpea—Mosaic
Cranberry—False blossom
Crocus—Mosaic
Crucifers—Mosaic
Cucumber—Mosaic
Currant—Reversion or nettlehead
Daffodil—Mosaic, yellow stripe or gray disease
Dahlia—Dwarf or stunt
Dahlia—Mosaic
Dahlia—Ring spot
Dahlia—Spotted wilt
Delphinium—Coarse etch
Delphinium—Stunt or witches'-broom
Dodonae—Spike disease
Eggplant—Mosaic
Fig—Mosaics
Freesia—Yellows
Geranium—Crinkle mosaic
Gladiolus—Mosaic
Gloxinia—Spotted wilt
Grape—Leaf roll

Grape—Mosaic
Grape hyacinth—Mosaic
Helichrysum—Yellows (aster)
Hippeastrum—Mosaic
Hop—Chlorotic disease
Hop—Mosaic
Hop—Nettlehead
Horse bean—Mosaic
Hyacinth—Mosaic
Iris—Mosaic
Jack bean—Leaf curl
Jack bean—Mosaic
Jimson weed—Mosaics
Jimson weed—Spotted wilt
Jobs'-tears—Streak (corn)
Legumes—Mosaic
Lettuce—Mosaic
Lettuce—Spotted wilt
Lettuce—Yellows
Lily—Mosaic
Lily—Yellow flat or rosette
Lily of the Valley—Mosaic
Lima bean—Mosaic
Loganberry—Dwarf
Loganberry—Mosaic
Lupine—Mosaic
Lupine—Spotted wilt
Marigold—Yellows (aster)
Melon—Mosaic
Mustard—Mosaic
Narcissus—Mosaic
Nicotiana—Aucuba mosaic
Nicotiana—Mosaic
Nicotiana—Spotted wilt
Ocean spray—Witches'-broom
Okra—Mosaic
Onion—Yellow dwarf
Pansy—Curly top
Papaw—Bunchy top
Papaw—Curly leaf
Passion vine—Woodiness or bullet disease
Pea—Mosaics
Pea—Streak
Peach—Little peach
Peach—Mosaics
Peach—Phony disease
Peach—Red suture
Peach—Rosette
Peach—Yellows
Peanut—Mosaic
Peanut—Rosette
Pelargonium (*see* Geranium)

Peony—Mosaic
Peony—Ring spot
Pepper—Calico or infectious chlorosis
Pepper—Leaf curl
Pepper—Mosaics
Pepper—Spotted wilt
Petunia—Leaf roll
Petunia—Mosaics
Petunia—Ring spot
Phlox—Mosaic
Physalis—Bunchy top
Physalis—Leaf roll
Physalis—Mosaic
Pineapple—Yellow spot
Plantain—Bunchy top
Plum—Mosaic
Plum—Plum pox
Poinsettia—Leaf curl
Pokeweed—Mosaic
Potato—Leaf roll
Potato—Mosaic (for others *see* special treatment of Potato mosaic)
Radish—Mosaic
Raspberry—Leaf curls
Raspberry—Mosaics
Raspberry—Streak
Rhododendron—Mosaic
Rhubarb—Mosaic
Rice—Stunt disease or dwarf
Rose—Infectious chlorosis
Rose—Wilt or dieback
Salsify—Yellows
Sandal—Leaf-curl mosaic
Sandal—Spike disease
Schizanthus—Spotted wilt
Sesame—Leaf curl
Sincamas—Mosaic
Sisal hemp—Mosaic
Snapdragon—Infectious chlorosis
Soybean—Leaf curl
Soybean—Mosaic
Spinach—Blight or mosaic
Spinach—Curl disease
Spinach—Curly top
Sorghum—Streak (corn)
Squash—Curly top
Stocks—Breaking
Stone fruits—Mosaic
Strawberry—Crinkle
Strawberry—Witches'-broom
Strawberry—Xanthosis or yellows
Strawberry—Yellow-edge disease

Sugar beet—Curly top
Sugar beet—Leaf curl or crinkle
Sugar beet—Mosaic
Sugar beet—Yellowing disease
Sugar cane—Dwarf disease
Sugar cane—Fiji disease
Sugar cane—Mosaic, mottling or yellow-
    stripe disease
Sugar cane—Sereh disease
Sugar cane—Streak
Sunflower—Mosaic
Sweet clover—Ring spot
Sweet pea—Mosaic
Sweet potato—Mosaic
Tea—Witches'-broom?
Tobacco—Crookneck
Tobacco—Etches
Tobacco—Leaf curl, Kroepock or streak
Tobacco—Mosaics
Tobacco—Rotterdam-B disease
Tobacco—Stunt (Delphinium)
Tobacco—Veinbanding
Tobacco—Witches'-broom
Tomato—Aucuba mosaic

Tomato—Big bud
Tomato—Bunchy top
Tomato—Delphinium stunt
Tomato—Fern leaf
Tomato—Leaf roll
Tomato—Psyllid yellows
Tomato—Spotted wilt
Tomato—Streak
Tomato—Tip blight
Tomato—Tobacco mosaic and other
    tobacco viruses, except ring spot
Tomato—Western blight, yellows or
    curly top
Tomato—Witches'-broom
Tomato—Woodiness
Tulip—Breaking
Turnip—Mosaic
Vegetable marrow—Mosaic
Watermelon—Mosaic
Wheat—Mosaic or rosette
Zinnia—Curly top (Beet)
Zinnia—Leaf curl
Zinnia—Spotted wilt

New hosts and new viroses reported since the compilation of this list are not included.

CHAPTER XVIII

DISEASES DUE TO UNFAVORABLE SOIL CONDITIONS:
DEFICIENCIES OR EXCESSES OF FOOD MATERIALS,
SOLUBLE SALTS OR WATER

DEFICIENCIES OF SOLUBLE SALTS

Considering the varying origin and composition of soils and the fact that plants must obtain the large part of their food materials from the soil environment, disturbances of nutrition due to insufficient amounts of some of the essential or accessory food materials may be expected under certain natural field conditions.

Chemical Elements Required by Green Plants.—Every green plant requires ten different chemical elements, and these must be obtained either in the form of elements or combined in compounds which are available in the environment in which the plant grows. The ten essential elements are as follows: carbon (C), hydrogen (H), oxygen (O), nitrogen (N), phosphorus (P), sulphur (S), potassium (K), calcium (Ca), magnesium (Mg) and iron (Fe). Most of the carbon and oxygen are obtained from the air as carbon dioxide and free or elemental oxygen and are used by the plant in the process of carbohydrate food manufacture, or photosynthesis, and in respiration. The great volume of elemental nitrogen which forms four-fifths of the atmosphere is available for only a few specialized plants which have the power to fix free nitrogen. Hydrogen is obtained in part from water. The four elements named are volatilized and lost when plant tissue is burned or reduced to ash, but the other essential elements P, S, K, Ca, Mg and Fe, together with a number of other accessory ones, are left behind in the ash. Crop plants, exclusive of the legumes, must get their nitrogen from the soil in combined form, and all crop plants must derive their ash constituents from compounds which are brought into solution in the soil water. Some of the nonessential elements of the ash serve no important function in the life of the plant, while others may be of value because of a stimulating effect or because their incorporation modifies certain plant structures.

The Uses of the Essential Elements.—Carbon, hydrogen and oxygen are combined to form carbohydrates, which are essential foods for crop

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plants. In addition to these three elements, nitrogen, sulphur and phosphorus enter into the composition of proteins and nucleoproteins which are manufactured by green plants and utilized in the nutritive process. *Potassium* is essential for healthy growth and accompanies and plays a part in carbohydrate synthesis. *Calcium* is necessary for normal leaf development; it exists as calcium pectinate, in the middle lamellae, which cement adjoining cells and may serve a protective action by combining with oxalic acid to form crystals of calcium oxalate which are insoluble. This would prevent the injurious effects from the accumulation of oxalic acid. *Magnesium*, if not an actual constituent, at least accompanies certain proteins and is contained in chlorophyll. *Iron* in minute amounts is essential for green plants, and its lack prevents chlorophyll formation. When deprived of iron, plants develop pale or chlorotic foliage. Some of the essential elements may serve in the growth of plants in other ways—for example, sulphur appears to have a stimulating effect on certain crops, while calcium appears to help in maintaining a proper soil reaction.

Elements Likely to Be Deficient.—Carbon, hydrogen and oxygen are generally available to the growing plant in sufficient amounts to satisfy the needs. In certain cases, lack of oxygen may cause asphyxiation of roots or play a part in storage troubles of plant products (see Chap. XIX). Water shortage probably causes injuries because of interference with other water functions, rather than by depriving the plants of hydrogen furnished. The principal deficiencies of chemical elements are of those supplied to the plant through soil compounds. Those which are most likely to be deficient in certain soils and to limit plant growth or give rise to abnormal or diseased conditions are nitrogen, phosphorus and potassium. Under certain conditions, sulphur, magnesium, calcium or iron may be lacking in the proper amount. The absence of an element or its presence in nonavailable form will lead to the same results. The shortage of a single one of the essential elements or of two or more in the soil of fields or greenhouses may simply retard or restrict growth and fruit formation if the shortage is not too pronounced; or if the deficiencies are greater, marked pathological conditions may result.

In addition to the ten elements generally recognized as essential in the nutrition, certain others which occur in the soil in relatively small amounts may have a corrective or therapeutic value for certain pathological conditions. The exact way in which this corrective effect is accomplished is in many cases uncertain. Some of these "trace elements" such as boron, copper, manganese, zinc and others have been used effectively for the correction of certain diseases of our crop plants. Some of the trace elements occur in plant tissues so constantly that a physiological significance is suggested.

Nitrogen Shortage.—Some plants when deprived of nitrogen are able to grow to maturity and produce blossoms and fruit solely by utilizing the nitrogen that was already stored in the seed, but the aerial growth is very much restricted or dwarfed in much the same way as in water shortage or drought injury. In plants dwarfed by drought, the relative size of shoot and root system is approximately normal, while, in the case of nitrogen deficiency, the root is abnormally elongated. It has been stated that this elongation of the root to many times that of the shoot is an infallible indicator of nitrogen shortage. It has been shown by cultures to be true for corn, and many cases of similar relations have been observed in plants growing in nitrogen-deficient soils under natural conditions. In pronounced nitrogen deficiency, the foilage may assume a light-green to yellowish-green color and with chronic or continued shortage become dry and yellowish brown. *

In many cases, nitrogen shortage is expressed only by a lowered quality of the commercial product, or in more pronounced shortage by dwarfed plants, and lessened production. The shortage of available nitrogen may cause modified flower development and consequent unfruitfulness. It is one of the factors that influences sterility and is also a factor in the premature dropping of fruit. It is also the belief that biennial or irregular bearing in fruit trees is largely a nutritional problem in which a disturbed nitrogen relation is the most important factor, although certain varieties are more prone to the habit than others.

The frenching or strap-leaf disease of tobacco is characterized by the production of long narrow, ribbonlike or strap-shaped leaves, with wavy or crinkled margins, dwarfing of the entire plant and, generally, showing more or less chlorosis, although in some cases this symptom may be lacking. The suggestion was first made by Valleau and Johnson in 1926 that frenching is a nitrogen-deficiency disease, and they and several other workers showed that it could be controlled by supplying available nitrogen. More recently it has been shown that the disease can also be controlled by several applications of a dilute solution of copper sulphate or aluminum sulphate, and that it can be produced by watering plants grown in sand with a water extract of field soil. It has been concluded (Shear, 1933; Spencer, 1935) that frenching is caused by some toxic principle that develops in some soils under definite environmental conditions.

The yellow berry of wheat is a specific trouble in which there is a disturbed nitrogen nutrition (see Special Treatment, page 487).

Phosphorus Deficiency.—While the majority of soils are not sufficiently lacking in phosphorus to produce pathological effects, the soils of some areas may respond to phosphorus fertilizers by increased production. This has recently been shown to be true for some areas in Montana. Although no field case of phosphorus deficiency with citrus has been

demonstrated, the symptoms of phosphorus hunger have been studied by soil, sand and solution cultures. Some of the effects produced were "fading of the chlorophyll, a burning of the blade in various locations, a reduction in leaf size, or a change to a dull brownish-green color without luster."

Magnesium Deficiency.—It has been shown that magnesium may be deficient in certain soils and under certain environmental conditions. Light sandy soils appear to be more likely to show magnesium deficiency than heavier soils especially if derived from rock formations originally low in magnesium. The conditions which lead to a magnesium deficiency are: (1) the removal of magnesium from the soil by continuous cropping and leaching from rather heavy precipitation; (2) the use of fertilizers that contain little or no magnesium for a long period; and (3) too heavy applications of potash on soils naturally low in magnesium. Marked magnesium deficiency symptoms were recorded following a rain of 7.92 inches in July (Wallace, 1941).

Fig. 207.—Leaves of tobacco affected with sand drown. (*After Garner et al., U. S. Dept. Agr.*)

The visible effect of magnesium deficiency varies from a mild or faint chlorosis to a pronounced chlorosis and in the more severely affected plants may cause internal or marginal necrotic areas or complete necrosis of the leaves, the symptoms developing progressively from the base upward. The chlorotic pattern follows the type of leaf venation showing

(1) in monocots like corn and other cereals as pale or chlorotic streaks between the parallel veins and sometimes a reddish discoloration of the margins and leaf tips; and (2) in many dicots as a rather characteristic mottle in which the chlorosis is confined to the intervascular areas, in the extreme types little green tissue remaining except the veins. This character is well illustrated by the leaf mottle of tobacco affected with sand drown, a disease shown to be caused by magnesium deficiency (Fig. 207). In some cases chlorosis appeared in the leaf margins, instead of in the intervascular areas. Spinach appeared to omit the chlorotic stage and develop at once papery white necrotic areas very similar to sunscald. In advanced stages on some dicots the leaves showed some cupping or curling, followed by necrosis and abscission. It is claimed that magnesium deficiency increases the susceptibility to frost injury.

In areas in which magnesium deficiency is common very pure forms of potash fertilizers should not be used unless supplemented with other material containing magnesia. The most economical method of providing the necessary magnesia is by the application of dolomitic or high-magnesium limestones, which are of additional value in correcting soil acidity; or some other product containing water-soluble magnesia may be used. Soils deficient in magnesia will need from 10 to 25 pounds of soluble magnesia (MgO) per acre depending on the crop to be grown. Help in maintaining the magnesium supply may be obtained by green manure crops such as rye, soybeans, grass, etc., which retard loss from leaching, and by the use of barnyard manure.

Potash Hunger.—Some plants require a larger amount of available potash than others in order that they may function normally. This is notably true of those crops which are manufacturing and storing large quantities of carbohydrate reserves. The large quantities of potash fertilizers used in certain sections are evidence of the beneficial effects of increased potash. The general effects of shortage of potash may be noted: (1) a reduced photosynthetic activity and consequently, a retarded or dwarfed growth of storage organs, such as fleshy roots or tubers, or in cereal crops the development of vegetative structures at the expense of the grains; (2) in woody plants a suppressed or weak development of terminal shoots which may end in a "dieback," as has been shown for both wild species and cultivated fruits; (3) the appearance of yellowish, brownish or whitish spots in leaves at first near the margin and later more general if the shortage continues and is pronounced; (4) the later blighting of the foliage and premature death if the shortage is not relieved. It has been shown that the amount of stored carbohydrates, such as sugars and starches, is in direct proportion to the amount of available potash. This will explain the weak shoot development of woody plants suffering from potash hunger, since the cellulose which must be formed

in.the construction of new organs is also a carbohydrate. While die-back may be the end result of a potash shortage, it should be remembered that various other factors may bring about a similar result. The effect of potash shortage can be demonstrated by growing seedlings in a potash-free substratum. After the reserve carbohydrate has been used up, no more can be constructed and growth ceases; but with the addition of a potassium salt, carbohydrate manufacture is resumed and growth con-tinues. It may be noted that in nitrogen or phosphorus shortage the plant generally completes its vegetative period with the production of all organs, vegetative and reproductive, although it remains dwarfed, while in potash shortage the plant reaches nearer a normal size, but blossom and fruit production are either decreased or inhibited and a premature death is the most likely end result.

It has also been shown that the addition of potash fertilizers to soils increases their water-holding capacity beyond that of the same soil with-out the addition of potash. This being true, a soil short in potash might increase the chances of drought injury. It has been reported that root crops which are suffering from shortage of potash are less resistant to decay during rainy periods and that they wilt more readily during hot weather. On this basis, one of the values of a potash fertilizer may be the increased resistance to parasitic inroads.

Potash hunger was brought into special prominence in America by the use of a large amount of no-potash fertilizer as a result of the cutting off of the German supply of potash at the time of the world war. It caused much injury in potato and tobacco sections of the eastern United States where the use of a potash fertilizer had been an established prac-tice. The symptoms and effects of potash hunger of potato were: (1) a change of color of the plants during July from a normal healthy green to a peculiar bronze or yellow; (2) the wilting and drooping of the leaflets while the stems stood erect; (3) the drooping and wilting of plants nor-mally green, the stem not having sufficient strength to stand erect; (4) the appearance of discolored areas on various parts of the stems; (5) the formation of a dry, discolored, spongy area involving the stem at about the surface of the ground; and (6) the premature death which always followed these symptoms. This trouble seemed to be most in evidence on the poorer soils or on those which had been heavily cropped with insufficient attention to the use of fertilizers during the preceding years and was aggravated by moisture shortage. In potash starvation of tobacco, plants are more or less stunted, the leaves puckered or with an uneven surface due to the difference in the rate of growth of the veins and the intercostal areas. The peripheral growth is retarded, and, as a consequence, the margins of the leaves and especially the tips curve downward, giving the condition which the farmer calls "rim bound."

The affected leaves are also discolored, a chlorosis beginning at the tips and margins of the leaves and advancing inward and downward; the lower leaves show this discoloration first. This chlorosis is followed by the appearance of small dead spots, and, with progress of the malady, large areas die, especially along the margins, which frequently become ragged and torn, producing the so-called "rim fire." In tomato plants, potash deficiency causes inhibition of cambial activity, limits the development of cork cambium and causes premature death if fruit is present, owing to the killing of the growing points by the withdrawal of potash into the fruit. A leaf scorch of the apple in England, characterized by marginal burning has been associated with a shortage of potash, but sometimes with other contributing factors.

Therapeutic Value of Some Rarer Elements.—Without entering into a discussion as to whether such elements as boron, copper, manganese and zinc have a food value or simply serve as stimulants to unlock necessary activities, some specific illustrations may be cited of recognized pathological conditions which have been cured or greatly relieved by treatment with small quantities of the rarer elements of the soil.

During the last few years, a voluminous literature has accumulated as to the curative value of boron for the crown and heart rot of beets, a trouble which for many years was considered to be caused by a fungus, *Mycosphaerella tabifica*, and later by a combination of this parasite and a disturbed nutrition. Three factors which aggravate this disease are: high alkalinity, drought conditions and a sandy or gravelly soil of poor water-holding capacity. The crown and heart rot has been largely overcome by the application of 15 to 22 pounds of borax per acre, well distributed in the soil; part applied preferably just previous to seeding and the balance later.

The curative value of boron has also been demonstrated for the "reclamation or bog disease," a trouble characteristic of swampy heath soils in European countries. Oats, barley, wheat, corn, red clover, swedes and turnips are some of the crops affected; oats show a whitish-green color with increased stooling; barley, yellowing and marginal curling or rolling of leaves and empty heads; wheat, longitudinal white stripes on the leaves and heads rudimentary or lacking; corn, increased tillering and poor yield; clover, leaf pallor, dry brown spotting and marginal chlorosis, with flowers late or scanty; swedes and turnips show stunting, discoloration and shriveling of leaves, with yellowish white lesions spreading in from the margin. The curative value of boron has also been demonstrated for the brown heart disease of turnips and swedes, cork and drought spot of apples and the crack stem of celery. In numerous controlled cultures the withholding of boron has produced well-defined pathological symptoms. A recent compilation has recorded boron deficiency in 24 of the states on 20 or more crop plants (Purvis, 1939).

. A condition apparently similar to the reclamation or bog disease of Europe has been prevalent in various crops planted in raw peat soils of the Florida everglades. It has been shown that wonderful responses have been obtained by treating the soil with 30 pounds of copper sulphate per acre. More recently the curative value of copper treatments for the reclamation disease of Europe has been demonstrated. The failure of boron in the everglades tests was due apparently to the large amount of boric acid used per acre (68 pounds), this strength proving toxic rather than stimulating. In many other cases copper has been shown to have a beneficial effect on production whether applied to the soil or sprayed on the leaves. In some unproductive peat soils (North Carolina), the addition of copper sulphate has induced chlorosis, rather than having a beneficial effect. A group of somewhat similar diseases have been benefited by treatment of the tree or soil with zinc salts. The affected trees have been either greatly benefited or cured by supplying zinc sulphate by spraying on the foliage, introduction through holes bored in the trunk or by applications to the soil. Perhaps first prominence for this treatment has resulted from the curative effects on little leaf or rosette of apples and pears in California and Washington. Zinc treatments have also proved beneficial for the bronzing of tung trees in Florida, the pecan rosette in Arizona and the mottle leaf of citrus in California.

Deficiencies of available manganese and the resulting nutritional disturbances of certain crops have been reported from widely separated areas as a result either of natural conditions or from farm operations. The condition is frequently associated with calcareous soils in which manganese is mainly insoluble. Even when definite disease symptoms have not appeared, marked increases in production have been obtained by applications of manganese by either spraying or by direct applications to the soil. In manganese shortage marked chlorosis of the foliage is a symptom, with retarded growth, leaves reduced in size, blighting of terminal buds, and necrosis of leaves and defoliation. Some specific cases of manganese deficiency diseases are the Pahala blight of sugar cane, the gray speck disease of oats and manganese deficiency disease of beans, peas and other truck and forage crops when grown on slightly acid or alkaline peat soils. Some of these troubles at least are prevented by spraying or dusting with manganese sulphate or by soil treatments with manganese sulphate and sulphur.

As a result of cumulative evidence during the last few years there seems to be a general trend to the belief that boron, zinc, copper and manganese are essential in traces at least for the normal nutrition of most crop plants.

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EXCESSES OF SOLUBLE SALTS

In the previous section it was pointed out that our crop plants require 10 different chemical elements in order that they may make a normal or thrifty development. Seven of these elements are furnished from soil

compounds, and mingled with these essential food materials are various nonessential materials.

Natural and Acquired Excesses.—Food materials furnishing essential elements or compounds consisting of unessential elements or even containing toxic elements may be present in excessive quantities in the soil. It may be assumed that for each crop there is an optimum concentration of each kind of available food material at which the best growth is maintained; beyond this optimum, conditions become less and less favorable until, with a given concentration, disturbances result which may be expressed in the appearance of symptoms of disease, while maximum concentrations may lead to death. If we consider the origin of our soils, it must be at once apparent that many native or residual soils in their virgin state may contain excesses which may inhibit or retard growth or even, in spots, entirely exclude all forms of plant life. In addition to the natural excesses, the composition of land under cultivation may be modified by our agricultural practices, such as irrigation, cultivation or the use of fertilizers, so that natural excesses may be increased or new excesses introduced.

General Effects of Soil Excesses or Overnutrition.—Surpluses of available food materials are very frequently accompanied by an abundance of soil moisture, and the type of growth may be influenced by both the nutrients and the water supply. Certain types of growth are, however, directly traceable to overnourishment, the first effect of which is to be seen in an increased vegetative development, a deeper green than normal, more succulent tissues and a retardation or suppression of reproductive functions. Such overstimulated plants are frequently more sensitive to unfavorable climatic factors and fall an easier prey to some parasitic invaders. General overnutrition leading to an excessive accumulation of plastic substances in the plant in proportion to their utilization may lead to pronounced morphological changes. Among these changes may be mentioned *phyllody*, or the transformation of floral organs into leaflike structures; *petalody*, or the transformation of calyx bracts into petals; *pistillody*, or the change of stamens into carpels; *abnormal proliferation*, as illustrated by "rose kings," sprouted pears, doubling of composite blossoms and secondary heads of composites; *fasciation*, or the flattening or banding of cylindrical organs; and *spiralism* (see Symptoms of Disease). It should be pointed out that this excessive accumulation of plastic food materials is not always the result of overnourishment but that other factors may influence the utilization of plastic materials and lead to similar responses.

Special Surpluses.—The occurrence of specified surpluses in which some particular food element or compound or toxic elements or compounds lead to injuries may be noted. The essential chemical elements most likely to occur in excess are nitrogen and calcium and less frequently

aluminum, magnesium, potassium and phosphorus. Two surpluses of special importance are *alkali*, or the excessive accumulation of soluble salts (see Alkali Injury) characteristic of arid or semiarid regions; and *soil acidity*, a rather indefinite soil condition, common in humid regions, that is generally corrected by the addition of lime. Renewed emphasis has recently been given to the fact that "infertility in soils may as well be due to the presence of organic substances of biological origin inimical to proper plant development as to the absence of beneficial elements." These toxic compounds are not to be looked upon as excretions from normal roots but appear to be produced under abnormal soil conditions or are formed from the decomposition of crop residues under the influence of soil organisms. Nonessential elements may be either inert or toxic; in the former case they may produce injurious results by chemical inter-action, in the latter by direct effects upon the living substance.

Excesses of Nitrogen.—Nitrogen is probably the most active of the essential food elements in contributing to general overnourishment of plants. Under natural conditions, it is rarely present in sufficient quantity to cause injury to our crop plants, but the amount may be increased to the danger point by certain farm-cropping prac-tices or by the addition of excessive quantities of nitrogen-containing ferti-lizers. Some of the injurious effects of too much nitrogen are: (1) delayed maturity by stimulation of vegetative growth; (2) dropping of flower buds; (3) a succulent growth with poor me-

Fig. 208.—Niter burning of apple leaves. (*Drawn after Headden, Colo. Bul.* **155**.)

chanical resistance; (4) dwarfing or retardation of growth with quantities too great for stimulation; (5) chlorosis of foliage, followed by burning or necrosis; (6) browning or corrosion of the roots; (7) gummosis and die-back in citrus or stone fruits; (8) lowering of quality in fruits or cereals; and (9) increase of susceptibility to infectious diseases.

Bud drop of sweet peas and roses may be induced by an excess of nitrogen but is increased still more if temperature and light relations are also unfavorable. Dwarfing, chlorosis and burning have appeared in cultures of sweet peas in which the amount of available nitrogen was ten times that of a rich soil of the same type. The so-called "niter poison-ing" or marginal burning of apple leaves in Colorado has been attributed to a surplus of soluble nitrates in clean, cultivated orchards due to pro-nounced activity of the nitrifying bacteria. The soils of bare or nearly

bare areas, so-called "niter spots" in sugar-beet fields (Fig. 209) in Colorado have been shown to yield 41.86 per cent of nitrates in the water-soluble portion (Headden, 1910).

Lime or Manganese Chlorosis.—It has long been known that certain plants, when grown on calcareous soils, develop a sickly chlorotic foliage. Some specific illustrations are the chlorosis of cultivated plants and native vegetation in the cretaceous soils of the southern Mississippi Valley, severe chlorosis and dwarfing of grapes in the vineyards of France, moderate or severe chlorosis of pineapples and sugar cane in Puerto Rico, chlorosis of coniferous seedlings in the forest nurseries of Nebraska and Idaho, of various fruit and nut trees in commercial culture in California

FIG. 209.—A niter spot.   (*After Headden, Colo. Bul.* **155.**)

and of pineapples on the black manganese soils of the Hawaiian Islands. All the above cases occurred on soils normally high in lime or manganese, but injury to citrus trees in Florida has been reported by the application of ground limestone as a corrective for soil acidity.   In many cases of lime chlorosis, the wood does not mature properly and the affected plants are more susceptible to winter injury.

In many cases the calcareous or manganiferous soils cause chlorosis by iron starvation.   Sufficient iron is present but in lime chlorosis the excess of lime changes the iron compounds into insoluble, colloidal iron which cannot be utilized by the plant, or in the manganiferous soils to a much more difficultly available ferric iron.   Some studies in pineapple chlorosis on manganiferous soil have led to the belief that there is a greater immobility of the iron in the plants owing to greater assimilation of lime caused by the excess of manganese.   In certain cases lime is not a corrective, and the chlorosis is attributed to a disturbed or inhibited iron

metabolism. Lime-induced chlorosis has been corrected in certain cases by the addition of boric acid (Truninger, 1938).

In the chlorosis of the first type, relief is obtained by supplying additional iron; while in the second type in which iron was not a corrective, the addition of small quantities of manganese salts restored the affected plants to their normal green color. Iron may be supplied in two ways: (1) By mechanical application on or in the plant. Two methods of introduction into the plant have been used: (a) by spraying the plants with a solution of an iron salt; (b) by injecting the iron salt into the wood of trees. (2) By adding iron to the soil, or by soil treatments to render the soil iron available.

Spraying with iron sulphate has been successfully used in forest nurseries and for the control of chlorosis of pineapples, but for fruit trees the injection method has been more successful, the best results following the use of ferrous and ferric citrate. In adding iron to the soil best results will be obtained if the soil is not too strongly alkaline, but for alkaline soils it is desirable to improve the soil conditions by modifying the soil reaction to an acid one or at least less alkaline. Barnyard manure, ammonium sulphate or sulphur are beneficial. The first, plus the addition of iron and aluminum sulphate, equal parts, at the rate of 1 pound for each inch of diameter of the plant, has been recommended (Crawford, 1939).

Soil-acidity Malnutrition.—Acidity as applied to soils generally indicates a condition that can be corrected by the addition of lime. Soil acidity is common mainly in regions of abundant rainfall, while alkali soils are confined largely to the arid or semiarid regions. The acidity of soils may result from the interaction of the residual components of the soil from cultural or cropping practices or from the leaching from heavy rainfall. The continued use of acid mineral fertilizers like acid phosphates or sulphur or sulphur salts that are oxidized to form acid may lead to detrimental soil acidity. The annual use of acid phosphate and other fertilizers in certain truck crop areas of Virginia increased the soil acidity until it became a limiting factor in the growth of truck crops. Injury to cotton has been reported from acidity following excessive applications of sulphur. It is a well-known fact that the acidity of the soil has a marked effect upon the character and distribution of native vegetation.

The first effect with moderate acidity will be *retarded growth* and a *pallor* or less intense green than normal, which if the unfavorable conditions continue may become more pronounced. Foliage may become *mottled*, showing lighter-green areas between the veins, or the *chlorosis* may become diffuse or general. Such affected plants may weaken and die prematurely, or growth may be resumed if rains modify the

acidity later in the season.   Roots of affected plants make a poor develop-
ment, and many of the lateral feeders may be repeatedly killed back.
It is a significant fact that certain soil organisms are distinctly favored by
acid conditions, for example, the pathogen of clubroot of cabbage.

The injurious effects of acid soils may be accounted for by: (1)
unfavorable hydrogen-ion concentrations; (2) direct effect of toxic ele-
ments, such as aluminum or magnesium which appear to occur in active
or soluble form when the soil reaction or hydrogen-ion concentration is
beyond neutrality; or (3) the nonavailability of certain essential nutrients
or their reduction in amount, or the elimination or suppression of the
supporting or auxiliary action of nutrients, so that normal absorption
does not take place.   The shortage of calcium may operate in two ways:
(1) by depriving the plant of the required amount; and (2) by preventing
the absorption and use of other necessary elements like potassium.   Acid
soils are frequently short on nitrogen or even phosphorus; they also
interfere with the process of nitrification and the establishment of
nitrogen-fixing legumes.

The acidity of a soil may be corrected by the addition of compounds
which will furnish the necessary bases to combine with the acids.   Cal-
cium is generally selected because it is cheap and effective.   Potassium
is too expensive, and magnesium may sometimes be harmful.   Agri-
cultural lime in quantities greater than 1 ton per acre is seldom economical
but up to 2 tons may be used.   Three different forms are available:
carbonate of lime (limestone); hydrated lime (slacked lime); and oxide
or unslaked burned lime.   Their relative lime equivalents are 1, $\frac{2}{3}$ and
$\frac{1}{2}$.   It is perhaps best to use the minimum amount required for a given
soil condition and employ other practices to build up and maintain
general fertility.

Boron Injury.—Boron in small amounts may be beneficial but is
extremely toxic to plants if the concentration is increased.   Boron
injury has been caused by: (1) the use of manure treated with boron-
containing larvacides for the killing of house flies; (2) the use of com-
mercial fertilizers which contain borax as an impurity; and (3) the use
of boron-containing irrigation water or of soils with a high content of
residual boron.

The effect of boron varies with its concentration and with the soil
and moisture conditions.   Some of the effects are: (1) retardation or
prevention of germination; (2) death of plants or stunting so as to give
imperfect and uneven stands, with much variation in the size of plants
of the same age, both tops and roots being affected; (3) absence of normal
color marked by the bleaching or yellowing of normally green parts,
especially the margin and tips of leaves, followed by tipburn in the more
severe types of injury; (4) reduced growth and premature ripening, with

lessened yields. The injury may vary from slight disturbances which are largely outgrown to complete failure of a crop when large amounts of borax are applied.

Boron injury was brought into special prominence by the use of potash salts from Searles Lake, California, when the Franco-German War excluded the standard German potash. The injurious effects of boron

Fig. 210.—Borax injury of potatoes. *A,* potato field showing injury due to use of a potash fertilizer carrying 33 pounds of borax per ton. A no-potash fertilizer used on field in the background; *B,* potato seed pieces from apparently missing hills, showing various stages of borax injury. (*Taken from U. S. Dept. Agr. Circ.* **84.**)

were studied especially on potatoes and beans in Maine, on corn in Indiana, on cotton and tobacco in North Carolina and on citrus and other tree crops in California. In the various studies injury was reported to some of the more sensitive crops by as little as 3 to 4 pounds per acre and severe injury by no more than 15 to 20 pounds per acre. Some of these results are difficult to explain in the light of more recent studies on the curative value of boron for certain diseases (see Deficiencies of Soluble Salts). The degree of injury appears to be influenced by the

time and manner of application with reference to seeding or planting, by the amount of soil moisture and by the actual concentration of the boron salts in the soil water. Leaching of borax by rains prevents a residual effect, as proved by planting tests which have shown that the toxic effect does not persist to the next season.

A number of theories have been advanced to explain the toxic action of boron: (1) that it is *antizymotic*, that is, that it prevents the formation and action of enzymes or ferments which are vital to the process of germination and growth; (2) that it interferes with the translocation of carbohydrate food by forming chemical union with sugars and related compounds; and (3) that it interferes with the formation of chlorophyll by withholding iron from the tissues and so induces chlorosis. Whatever its mode of action, it seems that boron in sufficient concentration may exert a profound influence upon the nutritive processes and the regeneration of the chlorophyll.

Since impure fertilizers are the source of borax in sufficient concentration to cause crop injury, protection lies in the use of fertilizers in which the borax content is below the danger point for the amount of fertilizer that is ordinarily used per acre. Past experiences have resulted in commercial concerns giving greater attention to putting pure, high-grade products on the market. The recent investigations in California suggest that some consideration may need to be given to the purity of the irrigation water. Boron toxicity in cultures was somewhat relieved by the addition of various amounts of ferric sulphate. Leaching appears also to overcome the toxicity.

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DEFICIENCIES OR EXCESSES OF WATER

Before considering the ways in which a disturbed water relation may affect the growth and production of crop plants, a brief outline of the function of water in the life of plants may be presented.

The Function of Water.—The uses of water are as follows: (1) *a solvent for and vehicle of transport of food and food materials* from the soil

into the plant and from cell to cell throughout the plant body, thus constituting 80 to 90 per cent by weight of the active plant cell; (2) as one of the crude materials (water plus carbon dioxide) for carbohydrate food manufacture, or *photosynthesis*, supplying the hydrogen and oxygen of sugars and starches which are later used for food and also taking part in other necessary chemical processes, such as the hydrolysis of the complex foods like carbohydrates, proteins and fats; (3) makes possible the *maintenance of turgidity* or a hydrostatic pressure within living cells, a condition which is necessary for, or essential to, growth; and (4) supplies transpiration or evaporation—the loss of water through aerial parts—and thus promotes and regulates growth. Water loss by transpiration is a measure of growth and the accumulation of dry matter in our plants. The internal structure and the external form of plants may be profoundly altered by the variations in the water relations either by the soil moisture or by the humidity of the air. A detailed consideration of such alteration would carry us into the province of plant physiology and ecology, but it must be evident that excess or dearth of water will so affect nutritive or other physiological processes as to produce either disease or death of cells, tissues, organs or entire individuals.

General Effects of a Disturbed Water Relation.—The demands of our crop plants for water are exceedingly variable, and even certain varieties of a species may be much more sensitive to moisture fluctuations than others. A plant may be provided with too much or too little water, or water may not be available at the right time. The health of a plant is affected by the moisture supply of both its soil and air environment, and the amount needed for a normal or thrifty development is influenced by various environmental factors, such as temperature, sunshine and physical properties of the soil. The effects of a water shortage will vary in the case of sudden and acute shortage or in chronic deficiency of water throughout a long period. The first marked response to a pronounced water shortage is wilting, the drooping of succulent shoots and the rolling of leaves. The loss of water faster than it can be brought up from the root system decreases the turgor of the cells, and the structures, normally tense or rigid, become limp and flaccid. This *physiological* wilting is a common sight on a hot summer day, but the wilted structures again become rigid during the night or when evaporation is retarded, and thus growth proceeds with only temporary checks. Marked water shortage will result in retardation of growth and dwarfing. Moisture deficiency rarely operates as a single factor, since high temperatures and intense sunshine are frequent accompaniments. A plentiful water supply stimulates growth and results in the production of succulent tissues as contrasted with the firmer structures of plants supplied with moderate amounts of moisture.

Effect of Moisture Deficiency.—If the shortage of moisture is not relieved, drought response in foliage is marked by yellowing, reddening or other discolorations, followed by leaf fall in woody plants. In plants suffering from drought, dead brown areas may appear in the intercostal areas of leaves, in the center of areolae of these areas, or the leaves may be blighted or burned at the margins or tips. It should be remembered that other factors, such as toxic substances acting internally or externally or intense light and heat, may give rise to somewhat similar symptoms.

Moisture shortage by its interference with nutritive processes may lessen the production and storage of reserve food. Tuber or root crops will remain small, and cereals will produce shriveled grains. Fruits may be spotted, deformed or under normal in size, or they may shrivel and fall prematurely. In woody plants, drought injury may not be evident entirely during the season of low moisture, but the effect may be delayed until the next season, when weak shoots may be formed or twigs or branches die back, producing *staghead* or *dieback*.

The transplanting of herbaceous plants or of nursery trees frequently causes a disturbance in the water relations that may result in either death or a retarded growth. If herbaceous seedlings are grown in the moist atmosphere of a greenhouse, hotbed or cold frame, the cuticle is delicate, the external epidermal wall thin and the tissues in general poorly suited to withstand the rapid transpiration of a dry air. Hence if such plants are suddenly transplanted to the field, they may wilt so rapidly that death will result. If the root system is broken or mutilated, the danger of injury is greatly increased. The reduction of loss in transplanting may be accomplished by: (1) the hardening of plants by gradually subjecting them to conditions which approximate those of the field, rather than by a sudden change; (2) care in preventing the multilation of the root system; (3) cutting back the top or removing leaves to bring about a balance between transpiration and absorption until the root system can provide the necessary water; or (4) the protection of the transplanted plants from the direct rays of the sun or from the force of the wind so as to retard transpiration until the plants have become established. Because they are not able to adjust themselves to the new conditions, many delicate plants of the greenhouse, if moved to the dry air of living rooms without being hardened, may wilt, drop their leaves and even succumb.

Some Effects of Excess Moisture.—The injurious effect of water-logged soils has been pointed out (see Diseases Due to Improper Air Relations), and the importance of soil oxygen in the life of our crop plants emphasized. It has been shown that, in addition to the phenomena of yellowing and decompositions connected with an oversupply of water in the soil, there is an actual decrease in production. Attention

has recently been directed to the fact that any effect on the plant resulting from driving out the soil oxygen or the prevention of aeration may be due to either the lack of oxygen or the excess of carbon dioxide which cannot be carried away acting directly on the plant or on the soil organisms. It is significant that water cultures of corn failed to respond to aeration of the nutrient solution.

An abundant water supply produces a type of growth that is more susceptible to the inroads of either bacterial or fungous pathogens (see Fire Blight) and is more sensitive to extremes of heat or cold. This may be illustrated by the *sunscald* of the potato. When a period of warm rain, which checks evaporation and supplies abundant water, is followed by a period of high temperature and bright sunshine, the leaf tissues which are suffused with water are frequently injured, and a sudden blighting of leaflets or portions of leaflets results. This condition is sometimes mistaken for the invasion of a parasite. The same temperature and illumination would have no injurious effect on tissue having a normal water relation.

The greater delicacy of the cell walls in plant structures provided with an abundance of water and the increased rate of growth frequently result in the rupture of organs, such as fleshy roots, tubers, stems or fruits. Such ruptures are initiated when an unusual supply of water is given suddenly, especially following a previous dry period. This behavior is well illustrated by the cracking of carrots, kohlrabi, beets, turnips, potatoes and even some herbaceous stems. In the potato, the rupture is frequently internal and then the condition known as "hollow heart" results, in which a central cavity is formed, bordered by brown, oxidized tissue. The rupturing of nearly mature, soft-skinned fruits, such as cherries, plums or tomatoes, when a rain follows a rather prolonged dry period is caused by high sap pressure.

The knotlike or pustulelike enlargements on various organs, such as stems, leaves or fruits, produced by the enlargement of groups of cells, are known as *intumescences*, while more extended swollen areas in which similar tissue changes have taken place are characteristic of dropsy or *edema*. An edema of the tomato has been attributed to an excess of water in the soil combined with insufficient light and improper temperature relations. Many theories have been proposed to explain the formation of intumescences and similar deviations from the normal development, but these cannot be given a detailed discussion here. Suffice it to say for our present purposes that they represent a disturbed nutrition of the cells in which the cell walls remain relatively thin and the cells become distended with cell sap, frequently reaching many times their natural size. It may also be noted that intumescences have been produced experimentally by either chemical or mechanical stimulation.

"*Bitten*" or perforated leaves, in which the blades are very much shredded, torn or perforated by irregular openings, are closely related to the formation of "intumescentia" from the standpoint of origin.   The production of enlarged lenticels on potatoes or other underground organs, as the result of excessive moisture, is also similar to the formation of intumescences.   In the so-called "tan disease" the bark of either roots or aerial portions becomes more or less swollen in either localized or extended patches, and the outermost cork layers break or peel away.   The surface

FIG. 211.—Blossom-end rot of tomato in well-advanced stage, showing characteristic appearance.   (*After Stuckey.*)

beneath the blisters may show a whitish granular or even woolly appearance due to large numbers of loosely arranged cells, many of which may become more or less elongated.   These loosely arranged cells die and then, under dry conditions, appear as a dry, reddish-yellow, brownish-yellow or brown powder which may easily be brushed off.   This condition may be induced on the apple by severe heading back which prevents transpiration at a time when root absorption is active.   A somewhat similar type of cell formation is responsible for the "woolly streaks" in the cores of certain apples, and the tendency to form such streaks may be especially pronounced in particular varieties.

The fall of leaves, the shedding of blossoms, the dropping of fruits or the casting of twigs may sometimes result from a disturbed water

relation, either an excessive supply, a shortage or abrupt fluctuations, although various other nutritional disturbances may play a prominent part. Such troubles as June drop of fruits, failure of fruits such as grapes to set or, later, their shelling, the blossom drop of tomatoes or sweet peas and the shedding of cotton squares or bolls may be mentioned in this connection. Although there are many factors that play a part in reducing the set of fruit, excessive rains and prolonged humid conditions at blossoming time play an important role. Aside from the indirect effects of such conditions on the nutrition of the plant, rain washes pollen

FIG. 212.—Cross sections of tomatoes affected with blossom-end rot. (*After Stuckey.*)

down to the ground, restricts either wind or insect pollination, causes pollen grains to burst and also washes away stigmatic secretions which promote the germination of the pollen grains.

Some Special Diseases Caused by a Disturbed Water Relation.—A few of the more important diseases believed to be connected with a disturbed water relation will be given brief treatment.

Blossom-end rot of tomatoes is characterized by a dark green, water-soaked area at the stylar end of the fruit which soon changes to lead color or brown or even almost black and in the later stages may be flattened or even slightly sunken and nearly circular in outline. Under humid conditions the appearance of the lesions may be modified by the entrance of bacteria or saprophytic fungi, a dark sooty fungus appearing rather frequently. The disease has been increased in severity by spraying with Bordeaux, by excessive water, by heavy applications of

barnyard manure and by too little water at critical times in the development of the fruit; and lessened by a culture which induces a uniform, continuous growth with neither overstimulation nor depressing influences. Experiences with artificial fertilizers have given conflicting results, hence positive recommendations are unsafe.   Any factor restricting the rate of water absorption or greatly increasing transpiration favor the development of the disease.   No commercial varieties are immune, but marked variations in susceptibility are known and some relief may be obtained by the selection of the more resistant varieties or strains.   The disease is increased by full exposure to the wind, and consequently should be lessened by suitably placed windbreaks.

Black-end of pears or hard end is characterized by the appearance of russeting at the calyx end which soon turns black and spreads to form a more or less extended area around the eye, in extreme cases causing some cracking and even spreading halfway down the side of the fruit.   The discoloration is superficial, but the flesh is more gritty and unpalatable than normal fruit.   The lesions appear on maturing fruit in the orchard and may increase in severity in storage.   The Bartlett is especially susceptible, but other varieties may be affected with some modification of the effects.   The disease appears to be connected with the use of Japanese rootstocks which interfere with the movement of water from the rootstock to the scion.   Trees showing the disease one year generally continue to do so in succeeding years with minor fluctuations in severity. High temperatures and low humidity, with the resultant heavy demand on water, seem to aggravate the trouble.   Black-end fruits have been shown to be consistently more alkaline throughout the season but more so toward the end of the season when the number of affected fruits reaches the maximum.

Cork of apples shows as internal brown, dry, spongy or corky patches much more extensive than in bitter pit which may or may not be evident by external irregularities.   Hollows may be formed in larger dry-rot areas by shrinkage of affected tissue.   The disease may appear with or without reduction in size, depending on severity.   *Apple blister* is an extreme effect in which the dwarfed fruits are covered with brown, more or less circular raised spots, which may later crack and scale off, leaving the surface rough.   The variability of the symptomology and the study of the trouble in widely separated localities have led to the use of such names as malformation, dry rot, blotchy cork, corky core, York spot, punky disease, hollow apple, crinkle and confluent bitter pit.   The trouble has been especially prevalent in irrigated areas in soils with poor water-holding capacity and also in similar nonirrigated soils.

Drought spot or spot necrosis of apples and prunes has also been attributed to a disturbed water relation.   The trouble in apples is marked

by the formation of large, irregular, somewhat depressed water-soaked lesions, generally located toward the calyx end, which later become somewhat depressed and show a shallow layer of dead brown tissue below the spot. In prunes the trouble appears first as a watery spot beneath the skin, followed by the degeneration of the affected tissue with the formation of gum which may burst the skin and ooze to the surface. Recent studies in New Zealand, New South Wales and in British Columbia have shown that both cork and drought spot of apples can be prevented or very greatly reduced by adding boron to the soil in the form of borax or boric acid at the rate of $\frac{1}{4}$ to 1 pound per tree. The disease is, how-

Fig. 213.—*A*, cross section of King apple affected with cork, showing brown corky tissue near the core; *B*, section of a York Imperial, showing pockets and brown corky tissue beneath the surface depression. (*After Brooks and Fisher.*)

ever, increased by insufficient irrigation. It would seem an open question as to whether boron is simply a therapeutic or corrective agent or an essential nutritional element. Soil applications of either boric acid or borax at the rate of 30 pounds per acre have been reported to give perfect control of drought spot and corky core (McLarty *et al.*, 1937).

Hollow heart of potatoes appears to be induced by an abundance of moisture and food material which forces a more rapid growth with medullary browning causing an internal split or cavity accompanied by some oxidation of the ruptured tissues. Some studies (Wenzl, 1937) have shown 87.5 per cent in large tubers as contrasted to 7 per cent in small. Digestion of the starch is followed by the death of cells. Several claims as to the virus relation of the disease have not been substantiated, since the trouble is not transmissible, causes no decline in yield and produces no aerial symptoms (Wenzl, 1939).

Internal decline of lemons is characterized by an internal necrosis and destruction of the tissues, usually at the stylar end, frequently beginning

in the green stage or the *silver* stage, but becoming especially marked in the *tree-ripe or yellow* stage. The physiological factors favoring the trouble are: (1) hot dry weather causing heavy transpiration; (2) the pull of the foliage on fruit moisture; and (3) failure of roots to supply the needed moisture. Reduced by controlled irrigation, careful selection of soils and sites, judicious use of organic fertilizers and protection by windbreaks.

Straight head of rice is due to a disturbed water relation inducing slow development of the heads, which remain green longer than normal and stand erect when normal heads are drooping. The glumes or other parts of the inflorescence are aborted, distorted, or perfect, but complete flowers still remain sterile. The disease can be prevented by proper control of flooding or watering.

Water core of apples is characterized by the presence of glassy or watery areas in the flesh or pulp located near the core or extending out toward the surface. The glassiness results from the filling of the intercellular spaces with cell sap and develops in the fruit on the tree during the ripening period. Earlier reports attributed the trouble to an excessive water supply especially after periods of drought, but more recent studies have shown that excessively high temperatures alter the permeability of the protoplasm and thus permit the cell sap to fill the intercellular spaces. Some recent studies have shown the relation of leaf area to the development of the trouble, no water core developing in fruits provided with 10 leaves (Harley, 1939). Affected apples recover more rapidly in common than in cold storage. The disease will be reduced to a minimum in well-cared-for orchards.

White spot of alfalfa is a disease which is marked by the formation of two types of lesions: (1) localized spotting and (2) marginal injury. Both types may be so severe as to involve the entire leaf surface. An unbalanced water relation induced by irrigation is thought to be the principal cause, but heavy rainfall following drought may be a contributing factor.

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YELLOW BERRY OF WHEAT

This undesirable condition of matured wheat grain, called "yellow belly" or "yellow berry," is familiar to farmers, grain dealers and millers in many sections of the country. The deterioration of hard winter wheats caused by the appearance of yellow kernels has been the cause of special concern since the initial investigations published in 1905.

Symptoms and Effects.—Yellow berry is only in evidence in the threshed grain. It is characterized by the appearance in the normally hard, flinty wheats of light-yellow, opaque, soft starchy grains called "yellow berries." The normal grain is hard, flinty and translucent, while the grains affected with yellow berry will show in the extreme a dull white or yellowish tinge throughout. In lesser degrees of injury, the starchy portions may be small localized spots or streaks or involve one-half or more of the berry. When diseased kernels are cut transversely, the affected areas appear white and starchy instead of horny or flinty.

Grains affected with yellow berry show three pronounced deviations from the normal: (1) a modification of the structure and contents of the endosperm; (2) weight and specific gravity below normal glassy grains of the same variety; and (3) a reduced protein content.

The "yellow berry" kernels show larger and more numerous vacuoles in the endosperm, larger starch granules, a higher percentage of starch, and crush easier than the normal flinty kernels which have been shown

by analyses to contain at least 2 per cent more crude protein.   The amount of yellow berry varies with the variety, season and locality, and it ranges from none to as high as 80 or more per cent.   This defect lowers the grade or quality of wheat owing to the generally accepted belief that the presence of the starchy grains lowers the quality of flour that can be produced.   For this reason, wheat showing yellow berry sells at a lower' price than the hard, flinty wheats higher in gluten.   Millers in some sections of the country have referred to wheat affected with yellow berry as deteriorated wheat, thus expressing a detrimental effect.   It has been claimed that yellow berry reduces not only quality but quantity of flour because of greater mechanical difficulties in freeing the bran from the floury portions of the starchy grains.

Fig. 214.—Diagrams showing the varying amounts of starchy and flinty endosperm in yellow berry of wheat

Etiology.—Since the first reports of this trouble, various explanations have been offered for its occurrence.   The principal causes which have been suggested are: (1) climatic factors operating upon the grain while in the chaff, either during the last part of the ripening period or after cutting; (2) hereditary tendencies operating independent of environment; (3) disturbed nutrition due to unfavorable water or soil relations.

Some of the causes of yellow berry suggested by various workers have been: improper harvesting and curing; long exposure of the cut grain to the weather; late seeding and consequent late maturity; excessive moisture from rain or irrigation; none of these are now believed to have a direct causal relation, although they may have a modifying influence on the tendency induced by other factors.   The incidence of the trouble has been increased by the use of potash and phosphorus fertilizers and very greatly decreased or prevented entirely by the addition of sodium

nitrate to the soil. It seems probable that the determinative factor in the production of yellow berry in wheat is the ratio between available nitrogen (nitric nitrogen) and the available potassium. It would seem then that the lowering of the available nitrogen content through cropping, without using methods to restore the proper nitrate balance, is an important factor in the production of yellow berry. Wheat passes through three periods in its development: (1) a vegetative period up to the emergence of heads; (2) a period of translocation during which the head fills; and (3) a period of desiccation or maturing. Conditions favoring a greater relative abundance of carbohydrates during the period of translocation will tend to produce a lower protein content.

The effect of available nitrogen on the percentage of yellow berry is shown by the results obtained in dry-farming districts of the Pacific Northwest. In Oregon and Washington, early-plowed summer fallow has given little or no yellow berry, while later-plowed summer fallow has produced wheat with a high percentage of the trouble. In the early-plowed land, conditions are more favorable for the work of the nitrifying bacteria. Consequently, the amount of available nitrogen is increased and the percentage of yellow berry is correspondingly decreased.

Prevention.—In the light of present information, it seems that the most reliable method of preventing yellow berry or reducing it in amount is by following a cropping system or adopting cultural practices which will keep up the available supply of nitrogen. Although the application of sodic nitrate in amounts of 40 to 80 pounds per acre is a certain preventive, at least under certain conditions, its use in general wheat farming would probably not be justified. The following practices may be followed with profit not only from the benefit to be derived from the reduction in yellow berry but also from the effect on general maintenance of fertility and increase in yields: (1) the rotation of crops with the inclusion of a legume preceding wheat whenever possible, the exact rotation to be varied for different regions; (2) the use of summer fallow in regions in which legumes cannot be successfully grown, giving special attention to early plowing and sufficient cultivation to provide the conditions favorable for the activities of the nitrifying organisms of the soil.

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ALKALI INJURY

When one thinks of alkali in its relation to natural vegetation or to crop production, a picture is presented of barren soil either devoid of plant life or supporting only a sparse and dwarfed plant cover. This

picture represents the extreme of the alkali effect, which in many cases may be only slightly in evidence. As a purely chemical concept, the word "alkali" refers to substances having a basic reaction, but as applied to soils and the growth of some plants it refers to the natural accumulation of soluble salts in such concentration as to cause injury. The very substances which constitute the alkali salts may stimulate growth when present in the form of dilute solutions. It is, then, the concentration of these salts, rather than the kind or quality, that is primarily responsible for deleterious effects of alkali soils.

FIG. 215.—Alfalfa being killed by alkali brought to the surface by a rising water table. (*Photograph by F. J. Sievers.*)

The Composition of Alkali.—Soils that are alkali may include the chlorides, sulphates, carbonates, bicarbonates, phosphates and nitrates of the common bases sodium, calcium, potassium, magnesium and sometimes ammonia. The actual constituents in a given locality are, however, variable, but the three principal ingredients are: (1) sodium chloride, or common salt; (2) sulphate of soda, or Glauber's salt; (3) and the carbonate of soda, or sal soda. The chloride and sulphate of sodium and other bases may become concentrated at the surface of the soil and produce a whitish incrustation, characteristic of what is sometimes called "white alkali." These alkali spots are especially noticeable in semiarid lands and become most conspicuous during the dry periods. The carbonates of the bases, but especially the carbonate of soda, are capable of dissolving the organic matter of the soil. The solution formed and the surface accumulations are dark, hence the popular name of "black alkali." Both white and black alkali are injurious to vegetation, but the latter is much more destructive, as would be expected from the nature·of its action on soil humus.

Symptoms and Effects of Alkali.—The effects of alkali will vary according to the concentration and kind of the salts present and the resistance or tolerance of the plant to alkali salts. Some of the effects are: (1) failure of seeds to germinate; (2) death of seedlings after reaching a few inches in height; (3) retarded germination, the formation of sickly slender plants marked by chlorosis and early death without flowering or fruiting; (4) retarded growth, chlorosis and some foliage burning but fruit ultimately reaching maturity. Established shade or fruit trees may show a retarded growth and dwarfing, short terminal shoots, leaves fewer and smaller than normal, and frequently showing much

Fig. 216.—An alkali spot. Note the absence of a plant cover. (*After Harris.*)

chlorosis, burning or blighting of leaves at the tip or margins and premature leaf fall. In certain cases the foliage may become a brilliant golden yellow, while in other cases there may be a sudden wilting without the warning symptom of chlorosis. It should be understood that these symptoms are not diagnostic as very similar responses may be caused by other soil excesses or deficiencies, low temperatures, etc. Black alkali may cause a corrosion of the bark at the crown, resulting in the extreme in girdling very similar to the collar-rot type of winter injury, or both alkali and winter injury may operate simultaneously.

Plants in their native habitats that have become adjusted to saline conditions show a modified structure designed to diminish water losses or transpiration, including reduced size of leaves, cylindrical or spinous forms, dense hairy coverings, depressed or sunken stomata, or multiple palisade layers. When our crop plants are forced to endure alkali some modifications of structure result, but such changes are slow.

Etiological Considerations.—Alkali causes injury to our crop plants in a number of ways: (1) by the reduction, or the prevention, of absorp-

tion; (2) by a reduction of transpiration; (3) by toxic action upon the living cells; (4) by interference with the chlorophyll apparatus and the process of photosynthesis; (5) by corrosive action upon roots or stems in contact with the concentrated solutions, especially sodium carbonate; and indirectly (6) by affecting the physical properties of soils and their biological activities.

The total amount of the salts found in alkali lands varies from 0.1 to 3 per cent of the soil taken to a depth of 4 feet. The absorption of water by plants begins to diminish when the saline solution approaches 0.5 per cent and ceases entirely when it rises to 3 per cent, so it is evident that alkali soils may retard or even prevent absorption. With a high concentration of soluble salts water is withdrawn from the root cells, and complete plasmolysis may be the final result followed by death unless the osmotic balance is soon restored.

Alkali salts have a very marked influence on physiologic functions of the leaves. Minute quantities stimulate transpiration, but, when present in sufficient quantities to bring about modifications of structure, transpiration is much retarded, and consequently growth is less. Judging from the frequency with which chlorosis is the result of alkali, the retardation or slowing up of the photosynthetic activities is one of the injurious effects of alkali. The alkali chlorosis is apparently connected with the iron and calcium nutrition since the solution of both elements is prevented by black alkali.

Alkali salts affect crop growth indirectly by modifying the physical properties of the soil, causing the "freezing up." The first effect to be noted is the puddling of the soil or deflocculating of the particles, producing a compact condition which prevents the rapid rise of water, while the more active condition is seen in the firm, hard crusts which form on the surface of the soil, thus affording mechanical interference with plant growth. In the arid regions, particularly in sections of abundant alkali, hardpans are very likely to form. The physical effects of alkali on soils in leading to unproductiveness are stressed as being fully as important as the toxic properties of the salt solutions, and are well illustrated by barren alkali soils which are nontoxic.

Alkali soils have a marked effect upon the life and activities of soil organisms, especially the nitrifying and nitrogen-fixing bacteria, decreasing their activities. Just how much importance should be attached to the modification of the biological activities of the soil by the presence of alkali is still somewhat uncertain.

Resistance to Alkali.—The common field crops in the seedling stage show the following relative resistance to alkali: barley, oats, wheat, alfalfa, sugar beets, corn and Canada field peas. Among fruit crops grapes are the most tolerant (45,760 lb. per acre), and mulberry the most sensitive

(5740 lb. per acre), with olives, almonds, figs, oranges, pears, apples, prunes, peaches, apricots, and lemons intermediate in the order listed.

The tolerance of plants to alkali is well shown by the following list with concentrations at which they are likely to succeed:

Excessive alkali (above 1.5 per cent)—Salt bushes and salt grasses

Very strong alkali (1.0 to 1.5 per cent)—Date palm and pomegranate bushes

Strong alkali (0.8 to 1 per cent)— Sugar beets, western wheat grass, awnless brome grass and tall meadow oat grass

Medium-strong alkali (0.6 to 0.8 per cent)—Meadow fescue, Italian rye grass, slender wheat grass, foxtail millet, rape, kale, sorghum and barley for hay

Medium alkali (0.4 to 0.6 per cent)—Red top, timothy, orchard grass, cotton, asparagus, wheat and oats for hay, rye and barley.

Weak alkali (0.0 to 0.4 per cent)—Wheat, emmer and oats for grain, kaffir, milo, proso millet, alfalfa, field peas, vetches, horse beans and sweet clover (Kearney, 1911).

Prevention of Alkali Injury.—Alkali accumulation is characteristic of semiarid lands in which the high evaporation brings the alkali salts to the surface, while there is not sufficient precipitation to redistribute them in the soil. Excess of soluble salts is sometimes sufficient in long-continued ground culture under glass to cause crop injury. Irrigation favors the surface accumulation of alkali in semiarid regions, since alkali previously well distributed in the soil may be brought to the surface with the rise of capillary water and left as a surface incrustation when the moisture evaporates. This is what is known as the "rise of alkali." The purity of the irrigation water, that is, its content of alkali salts, is of importance, as the alkali accumulation will be accelerated by the rise of impure water.

No single practice will handle the difficulties encountered in cropping alkali lands. It may be noted first that lands favoring alkali injury need heavier irrigation than lands in which there is no alkali problem. Also that irrigation water carrying much alkali must be used more copiously than purer water. The principal methods of handling alkali lands so as to prevent crop injury or to reduce it to a minimum are as follows:

1. The use of alkali-resistant or alkali-tolerant crops. The use of a heavy alkali feeder such as sugar beets may make the land suitable for a more sensitive crop.

2. The adoption of cultural practices that will keep the alkali well distributed in the soil or retard or delay its accumulation at the surface such as: (a) cultivation to keep a surface soil mulch; (b) the use of a surface mulch of manure, straw, leaves or sand; (c) the establishment of a crop cover that will shade the soil; (d) the use of deep-rooting crops.

3. The burying of the surface soil by deep plowing.

4. The lowering of the water table and the prevention of seepage by the use of cement-lined ditches.

5. Removal or neutralization of the alkali by: (a) underdrainage by deep ditches alone or supplemented by flooding and leaching; (b) diking and flooding to a depth of several inches for some time, followed by good drainage and heavy applications of manure; (c) the neutralization of the sodium carbonate or "black alkali" by treatment of the land with either gypsum or elemental sulphur. The sulphur should be a finely pulverized brand and should be mixed with the soil by shallow plowing or disking. Some recent tests claim better results with a mixture of sulphur and gypsum than with either one alone. Even virgin black alkali lands can be reclaimed by chemical treatments supplemented by drainage and good irrigation.

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BITTER PIT

This disease has been described under a number of other common names such as "fruit spot," "apple brown spot," "spotted apples," "fruit pit" and "Baldwin spot." It should not be confused with a number of other spot diseases of the apple such as drought spot, cork, Jonathan spot, Jonathan freckle, scald and stigmonose.

Bitter pit was first recognized in Germany in 1869 under the name of "stippen," but it seems probable that it has affected the apple from the time when it began to be generally cultivated. At the present time bitter pit is recognized as a disease of apples wherever they are grown but seems to be most serious in certain irrigated areas, especially in America and Australia.

Symptoms and Effects.—The first external evidence of the disease is the appearance of slightly discolored spots on the skin of the fruit— darker red in red fruits and a deeper green on the yellow-skinned fruits. At first these spots are not depressed, but soon they become more or less sunken and assume the character of typical bitter-pit lesions. Such spots are more or less circular and vary in size from minute specks to others ¼ inch or more in diameter and appear like dents in the skin. They are not localized on any particular part of the fruit but are likely to be most numerous toward the calyx end and even in the most severe

cases are absent from a small area around the stem. Two or more pits standing adjacent may be confluent and so give rise to larger and more irregular lesions. The coloration of the skin as noted for the young spots persists for some time in the pits, but, finally, the depressions become brown owing to the death of both surface cells and underlying pulp tissue. The skin lining the pits remains unbroken throughout the course of the disease. The trouble may appear on fruit that is half grown, but generally it is not evident until approaching maturity and in many cases not until the early part of the storage period.

When an affected apple is cut in two, it is generally found to show internal groups or masses of brown necrotic pulp cells which show no

Fig. 217.—Bitter pit of apple.   (*After McAlpine.*)

connection with the external pits and the necrotic tissue just beneath them. The internal necrotic areas are generally more numerous in the peripheral portion of the pulp, but they may occur at any point outside the core wall. It is frequently noted that the fruits which show no external marks of the disease are already affected internally.

The pulp or flesh directly beneath a pit as well as the discolored spots in the interior consist of a mass of dead brown cells, dry and more or less corky or spongy in character. At first the dead tissue is a light brown, but later it becomes a darker brown, and generally has either a slight or a very pronounced bitter taste, which makes the name "bitter pit" especially appropriate.

Bitter pit may vary greatly in its severity and the damage which it causes. Apples may show only a few small spots, or the lesions may be as numerous as the pits on the face of a person who has suffered a severe

attack of smallpox, suggesting such names as "measles" and "smallpox." The affected apples are not destroyed, but they are of poor quality and also inferior in appearance. The disease is of special concern from its effect upon grade or pack, especially when so much attention is being paid to the production of extra-fancy fruit. A crop showing bitter pit at picking time is likely to suffer deterioration during storage even though carefully sorted and should not command the price of the best fruit. No accurate estimates of the actual losses from bitter pit are available, but it is certain that the disease is of world-wide importance and is taking a very considerable annual toll.

Fig. 218.—Section of an apple showing bitter pit and also moldy core. (*After McAlpine.*)

Etiology.—Bitter pit is a disease of definite and pronounced symptoms which permit its easy recognition after one really becomes familiar with its various expressions. In past time it has been attributed to bacteria, fungi, insects, mechanical injuries, degeneration from old age, varietal peculiarities, unfavorable grafting, shortage of boron, and to poisoning by the absorption of arsenical compounds or other spray materials through the skin. This last theory was later modified to the view that the toxic substances were absorbed by the roots and distributed through the natural channels, but this theory was not substantiated by other workers.

For some years bitter pit has been considered as a nonparasitic malady associated with a *disturbed water relation*, but there never has been any unanimity as to the exact way in which this disturbance operates. Regardless of the real cause of bitter pit, it should be of value

to understand the structure and functions of the normal fruit. The fruit has its own supply of vascular bundles, which enter through the fruit pedicel; these are distributed throughout the pulp, making a vascular network, and a very large number terminate in the peripheral portion of the fruit, which is protected by a suberized epidermis interrupted only by the lenticels. Water and mineral substances taken from the soil, and carbohydrates manufactured by the leaves, are carried into the apple and distributed to the pulp cells. The carbohydrate in the growing apple is mostly in the form of starch, but, as the fruit ripens this is converted into sugar. During the growing period of the fruit, transpiration is very active, and large amounts of water are passed out through

Fig. 219.—Late stage of bitter pit on Rhode Island Greening apple. (*After Brooks and Fisher.*)

the lenticels, just as water is lost from leaves through their stomata. Transpiration is not simple evaporation of moisture but is a physiological process which will be affected by supply of moisture to the root system, air humidity, air movements, temperatures, light, etc.

An examination of the groups of dead cells in bitter pit will show that they are always closely connected with certain branches of the vascular bundles. The individual cells are brown, devoid of sap, more or less collapsed and plasmolyzed but sometimes ruptured and contain starch grains that were not converted into sugar. This presence of starch in the affected cells has been taken to indicate that the changes which initiated the injury occurred before the ripening processes were completed. It has also been definitely proved that bitter-pit lesions, which first make their appearance after the fruit is in storage, are always located and the

injury started while the fruit is still on the tree. It may be noted that the lesions are located at the ends of water-conducting vessels, either at the periphery or internal.

Six main theories have been advanced to explain the killing and drying of the affected cells: (1) *the ruptured-cell theory,* which explains the killing of the cells as due to cell rupture from high sap pressure following dry conditions; (2) *the crushed-cell theory* explains the necrosis as due to the killing of cells whose starch transformation is backward by neighboring cells having a higher osmotic pressure resulting from their higher proportion of sugar in the cell sap; (3) *the starved-cell theory,* which attributes the death of cells to water shortage and failure to receive sufficient mineral food; (4) *the concentrated cell-sap theory,* which holds that the death of cells is caused by water loss which concentrates the acids and other constituents of the cell sap until a condition toxic to protoplasm has been reached; (5) *the plasmolyzed-cell theory,* the death of cells resulting from the withdrawal of water from the cells containing starch by those containing sugar; (6) *the virous theory.* This last explanation has been offered by Atanasoff and some other European workers who consider the bitter-pit symptoms but one phase or expression of a mosaic virus. The existence of a leaf mosaic of apple in Bulgaria and adjacent regions would seem established from published work, but the relation of bitter pit to this symptom rests largely on indirect evidence. The belief that two different diseases exist in the European areas is strengthened by the fact that two years of careful study by the writer and assistants in the irrigated areas of Washington, where bitter pit is common, have failed to find mosaic as a foliage symptom. More recently various writers have suggested a shortage of boron, but supplying this by dressings or injections has failed to give any relief. Since internal cork responds to boron, bitter pit must have an entirely different etiology.

Predisposing Factors.—Acceptance or rejection of a theory does not alter the fact that certain soil or climatic factors or cultural practices favor or promote the expression of the disease. According to various reports, bitter pit is favored by: (1) soils of poor physical or mechanical condition, especially those that are low in humus and have a poor water-holding capacity; (2) alternating wet and dry weather or very dry conditions followed by heavy rainfall, especially if this fluctuation comes relatively late in the season; (3) light irrigation during the early part of the season and late heavy irrigation; (4) heavy irrigation throughout the season, but less than by the variable light and heavy irrigation; (5) light irrigation throughout the season more than by heavy irrigation followed by light; (6) conditions which bring about the production of a light crop of large-sized fruit or of oversized fruit in general; (7) heavy pruning or a system of pruning which throws the fruit production onto the main

limbs rather than on well-distributed laterals and also heavy thinning; (8) fluctuating temperatures and humidity near the ripening period; (9) excessive transpiration or climatic conditions which cause a water loss out of keeping with the available supply. It has also been noted that apples on weak wood are more subject to the disease than those on strong branches. When conditions, whether natural or artificial, are such as to promote a uniform growth from early spring to the ripening period, the chances of bitter pit will be reduced to a minimum.

In stored fruit, the disease will not appear unless it was initiated during the growing period, but if the fruit is already affected, the disease will progress most rapidly if the storage temperature is relatively high and will also be favored by fluctuations in humidity and temperature. In other words, it will be favored by conditions which promote cell activity and retarded by conditions which delay or retard maturing or aging of the tissues, hence, by low temperatures. It is also stated that, in some cases at least, the disease is worse on early-picked fruit than on well-matured or late-picked fruit.

Varietal Susceptibility.—The apple is the crop most seriously affected by bitter pit, but it is recognized as a disease of pears and quinces. The different varieties of apples appear to show different degrees of suscepti- bility. Some are recognized as uniformly and severely affected, others as medium in susceptibility, while others are only slightly susceptible or almost immune. According to some reports, varieties very susceptible in one locality are reported less susceptible in another region. In North America the Baldwin is conceded to be a very susceptible, if not the most susceptible, variety, but, in some states of Australia, it has been reported as medium in susceptibility. The Cleopatra (Ortley) and Northern Spy pit very badly throughout all the states of Australia, and the latter variety is listed as among the most susceptible in America. The Stay- man, Black Ben and Grimes Golden are very susceptible, especially in the Pacific Northwest. Probably the great majority of our best com- mercial apples are either very susceptible or moderately susceptible to the disease, so that only partial relief can be expected from the selection of resistant varieties, but hope has been offered that pitproof varieties of high commercial value may be obtained by the crossing and selection of liable and nonliable varieties.

Control.—A considerable measure of relief from bitter pit can be obtained by the following practices: (1) The selection of known resistant varieties for planting. (2) The top grafting of established susceptible varieties to resistant varieties. Excellent results have been obtained in Washington by top grafting of such varieties as Stayman, Black Ben, etc. to either the common Delicious or one of the newer Delicious strains. (3) The handling of established orchards with careful consideration to

the following cultural practices: (a) try to increase fertility by suitable fertilizers and cover crops; (b) follow cultural practices to conserve moisture, maintain an even seasonal distribution and provide good aeration of the root system; (c) avoid crowding; (d) guard against light settings coupled with extra-vigorous vegetative development; (e) prune so as to secure an evenly distributed crop but avoid heavy or excessive pruning; (f) thin so as to produce fruit of moderate and uniform size, rather than small and large; (g) avoid overirrigation or underirrigation and especially heavy, late irrigation; (h) avoid too early picking, or too late picking. Prime maturity will give the best results.

With the recognition that bitter pit will become more pronounced during storage provided the fruit was affected at the time of picking, several courses are open to prevent or reduce loss: (1) early marketing to bring the fruit to the consumer before the fruit lesions have developed; (2) holding the fruit under conditions of high relative humidity (80 to 90 per cent) and as near as possible at temperatures of 30 to 32°F. during storage or transit to market; and (3) quick consumption after removal from cold storage. In some varieties immediate ripening at 70°F. has given less bitter pit than 50 or 32°F.

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CHAPTER XIX

DISEASES DUE TO IMPROPER AIR RELATIONS, HIGH TEMPERATURES, LOW TEMPERATURES AND UNFAVORABLE LIGHT RELATIONS

IMPROPER AIR RELATIONS

Living plants or plant structures stand in intimate relation to their air environment from which certain materials must be obtained and to which by-products of their activities are contributed. Unfavorable air relations may interfere with income and outgo of gaseous materials and thus induce disease.

General Air Relations of Plants and Plant Structures.—Every living portion of a plant body must be so related to its environment or to other plant parts as to obtain a supply of oxygen. In other words, every living part of the plant breathes. In this process of respiration, complex chemical changes are taking place within the living cells, but certain end products mark the process, carbon dioxide being given off and oxygen consumed. Oxygen is taken in through aerial parts and diffuses throughout an intercellular system of connecting spaces; it is also absorbed by the root system of the plant from the air in the spaces between soil particles. In the case of land plants, the roots cannot obtain their needed oxygen from or through the aerial parts but must obtain it direct from their soil environment. Respiration increases in the seed with absorption of water and exposure to favorable temperatures and continues as an active process throughout the life of the plant. In dormant plants or plant structures like tubers, rhizomes or fleshy fruits, etc., in which the life processes are at a low ebb, respiration does not cease until life is extinct. Any pronounced interference with the respiratory exchanges will result in lowered efficiency and, if continued, in evident ill-health and final death.

It is only under exceptional circumstances that the breathing of the aerial parts of our growing crops is likely to be seriously interfered with. A heavy deposit of dust or of inert particles such as cement dust may coat the aerial parts and choke the stomata of leaves and thus cause pronounced injury by interfering with the exchanges of gases. Dormant structures like tubers or bulbs or languid organs like fruits, when removed from their natural environment and collected in closely crowded quarters of the storehouse or the market package, may suffer from the lack of

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sufficient aeration. The oxygen requirement may not be satisfied, or the stagnation of the air may delay the removal of volatile products of protoplasmic activity.

Since underground parts must get their oxygen from the soil air, and since there are many ways in which the air content of the soil may be lessened or its free circulation impeded, asphyxiation of roots is a fairly common phenomenon. This may cause pronounced disturbances in the life of the plant or may end in its death. Most cultivated plants cannot obtain sufficient oxygen from water; hence a soil saturated with water will not supply sufficient oxygen to the root system. This will explain in the main the injurious effects of heavy, poorly drained or waterlogged soils, "wet feet," flooded lands or other conditions in which water drives out the soil air which is so essential to a healthy development. The physical structure of the soil may be such as to impede the circulation of the air so that the oxygen supply may be used faster than it is replaced, or the position of the root system may be such that new supplies of oxygen do not reach them rapidly enough. This will explain the injurious effects of closely compacted or hard soils, silt deposits, surface coverings of cement walks or pavements, deep seeding or setting of plants or filling around trees in grading for the construction of buildings. Seeds may fail to germinate, growing annual crops may sicken and die or lead a struggling life or trees may blight or die back because of the inability of the root system to obtain the necessary air.

Important Diseases Due to Improper Air Relations.—The importance of certain specific troubles caused by a disturbed air relation has justified special studies, and a few of the more serious of these diseases will be briefly considered.

Blackheart of potatoes, a disease appearing in stored stock or in field stock under conditions of abnormally high temperatures, is characterized by an internal necrosis, or blackening of the center of the tuber and sometimes of more external parts. Large tubers are more susceptible than small and are most susceptible during the middle of the dormancy period. The discoloration may start at the center, and, if the inciting conditions continue, the blackening may advance until it reaches the surface. A variation of this symptomology has been noted in the formation of shallow brown surface discolorations of varying extent, designated as *surface breakdown*, or "button rot." This may be followed by the internal discolorations of typical blackheart. Blackheart has been shown to result from an asphyxiation of the tissues of the tuber owing to lack of sufficient oxygen, and develops when the tubers are exposed to too high temperatures during storage or transit to market or to crowding in continuous high ranks of sacks or in bins of too large volume in poorly aerated storage rooms which permit an accumulation of carbon dioxide

and a depletion of oxygen in the surrounding air. The Indian Market variety is reported to be very tolerant to high temperatures.

Black leaf speck is a disease of cabbage, cauliflower and other crucifers which appears during storage and transportation to market. This is characterized by the appearance of small, sunken black specks, sometimes only on the outer leaves but, in the extreme, involving the heart leaves. This sometimes appears in cabbage left in the field until late, but it occurs mainly in the late winter on stock held in storage cellars, pits or in cold storage. It has been shown that black leaf speck will result when storage conditions are such that a constant supply of oxygen is not available and

Fig. 220.—Blackheart of potato.

sometimes also at low temperatures, near 0°C., despite the presence of an abundant supply of oxygen. Storage under conditions to permit a constant supply of oxygen, with the avoidance of too low temperatures, should prevent the development of this disease.

Red heart is a type of breakdown of lettuce and also cabbage characterized by the reddish coloration of the heart leaves, chestnut brown in head lettuce and a typical red color in cabbage. It has been shown by experimental tests that red heart is induced by restricted aeration or a limited supply of oxygen and is aggravated by soft rots in the outer leaves which makes gaseous exchanges more difficult. It does not seem to be quite clear as to why cabbage develops black leaf speck in one case and red heart in another, unless it is more likely to develop red heart if the asphyxiation occurs at temperatures somewhat above the temperatures of common cold storage (32 to 34°F.).

Internal browning of the Yellow Newtown apple is a defect that appears in cold storage as brown streaks in the pulp radiating outward from the center. The discoloration is believed to be due to the accumulation of essential oils or 'similar deleterious substances produced by changes in the apple tissue during storage. The disease is prevented in large part by picking at prime maturity and prompt storage at temperatures of 37 to 40°F. Ventilation in storage is of special value in preventing internal browning especially if it is necessary to store at temperatures below 36°F.

Brown heart of apples and pears is a functional disease characterized by a discoloration of much of the flesh between the core and skin without external skin changes. This disease has developed especially in shipments of apples from Australia to England, and it has been shown to result from insufficient ventilation of the ships' holds. Respiration of the apples caused the consumption of much of the oxygen and an accumulation of carbon dioxide, resulting in an asphyxiation of the fleshy tissues. The danger point was reached when the air contained 13 or more per cent of carbon dioxide. Delay between picking and cooling hastens ripening and increases liability to brown heart. If held at 38 to 44°F., the onset of the trouble occurs sooner than if held at 32 to 34°F. The trouble is prevented by a forced ventilation to prevent the carbon dioxide accumulation from reaching the danger point.

Pear scald and *core breakdown* are two closely related troubles: the first characterized in the early stages by a brown or black discoloration of the outer tissues of the fruit, and later by internal discolorations and off-flavors; the second by a brown discoloration and softening of the core tissues. Pear scald does not occur on fruit ripened at ordinary temperatures. It has been shown by Harley and Fisher (1927) that acetaldehyde accumulates in pear tissue when scald develops, and they consider this as the possible causal agent. This view is not accepted by later investigations (Thomas, 1931). It is shown that acetaldehyde accumulates progressively in the diseased tissue but that there is no evidence that it is formed in advance of the incidence of the disease, although it is admitted that it may aggravate the trouble after it has been initiated. In other words, the formation of acetaldehyde is simply an accompaniment of changes in ripening tissue rather than the cause of these changes. Late picking is conducive to the development of core breakdown, while early picking or storage before prime maturity favors scald.

Soft scald of apples is characterized by small brown spots from ⅛ inch or less across to large irregular areas that are frequently more or less transversely elongated, and it involves a certain amount of subepidermal tissue. It has been compared to the effects of touching or rolling an

apple on a hot stove. The margins of lesions are sharply defined, the skin tightly drawn and the affected tissue has a cooked appearance. This has been described by one writer under the name of "dry brown rot." Jonathan, Rome Beauty, Stayman Winesap and more rarely other varieties are susceptible to soft scald. A closely related trouble of Grimes Golden and Wealthy called *soggy breakdown* affects the cortical tissue for a depth of $\frac{1}{4}$ to $\frac{1}{2}$ inch and, in its earlier stages, is not evident on the surface. The discolorations may later involve a large part of the cortex. This is a disease of early storage and is entirely distinct from internal breakdown or senility necrosis. These two troubles are not prevented by the use of oiled wraps, but they have been greatly reduced by the Brogdex treatment and by immediate packing and cold storage. The tendency to soft scald and to soggy breakdown has been largely removed by a short prestorage exposure to carbon dioxide gas, and the earlier report as to the value of the Brogdex treatment for soft scald has been substantiated by successful control by coating the fruit with a 50-50 oil-paraffin mixture.

Some recent tests (Sutherland, 1936) showed a decrease of soft scald by storage at temperatures above 34°F. At this temperature 10 per cent of soft scald was produced as contrasted to none at 42°F. This author and others have recommended timely picking, immediate storage at 36 to 38°F. and provision for aeration and ventilation in storage.

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HIGH TEMPERATURES

The life of a plant is a complex of physiological processes which are operating according to chemical and physical principles: absorption, food manufacture, digestion of foods, assimilation, translocation of food, respiration, transpiration and minor life phenomena which lead to growth, maintenance of life and reproduction. All of these life processes are influenced by the temperature to which the plant is exposed, and this may be illustrated by the relation of temperature to growth. Life is possible only within the limits of certain temperatures, and growth has an even narrower temperature range.

General Temperature Relations of Plants.—There are three *cardinal points* in the temperature relations of any species of plant:

1. A *minimum* temperature or degree of warmth, at which growth first begins.
2. An *optimum* temperature, at which the growth is most rapid.
3. A *maximum* temperature, beyond which growth ceases.

As the temperature rises from the minimum, the growth is gradually increased up to the optimum, while, beyond the optimum, the growth becomes slower and slower until the maximum is reached and it ceases. Life may continue below the minimum, the plasma body passing into a *cold rigor,* but death may result if the temperature drops too low, that is, below the *subminimum;* or, again, the plant may survive above the maximum growth temperature, the living substance existing in a stage of *heat rigor,* but succumb if the temperature becomes too high or is maintained for too long a period above the supramaximum. Since every physiological process has its own cardinal temperature points, it must at once be apparent that thriftiness of growth and productiveness of a crop are influenced by the temperatures to which it is exposed. The various organs of a plant may have different cardinal points; hence fruits may suffer injury when vegetative parts escape unharmed, or flowers may be burned by temperatures that cause no injury to leaves.

Temperature is one of the factors in the climatic complex that plays a most important part in the natural growth and distribution of plants. In addition to being exposed to the vicissitudes of climate, crop or cultivated plants are subject to the intervention of man, and, consequently, they are frequently forced to attempt their life processes under uncongenial temperatures and other unfavorable environmental conditions. The temperature limits for growth lie, in general, between 0 and 50°C. Some growth may take place in certain plants at, or even slightly below, the freezing point of water, while some fresh-water algae that frequent hot springs may actually thrive at temperatures of 73°C. or slightly higher. Even our common crop plants show marked variations in their cardinal temperatures, as may be illustrated by the appended table of cardinal points of growth.

	Minimum, degrees centigrade	Optimum, degrees centigrade	Maximum, degrees centigrade
White mustard (*Sinapis alba*)............	0.0	21	28
Garden cress (*Lepidium sativum*).........	1.8	21	28
Barley (*Hordeum sativum*)...............	5.0	28	37.7
Wheat (*Triticum vulgare*)...............	5.0	29	42.5
Corn (*Zea mays*).......................	9.4	33.7	46.2
Squash (*Cucurbita spp.*)................	14.0	34	46.2

Types of Heat Injury.—The principal types of heat injury are: (1) retarded growth and undersize or failure to mature the flowers and fruit; (2) localized killing of tissues or a sunburn or sunscald of leaves, flowers or fruits; (3) localized killing of stem tissues or the formation of heat cankers; (4) defoliation or premature shedding of leaves; (5) premature ripening of fruits; and (6) death of the plant as the result of a general heat necrosis. It should, of course, be recognized that the high degrees of heat are frequently accompanied by intense sunshine and extremes of drought which intensify the injury. It has been shown that heat injury is due to poisoning by ammonia which accumulates in plant tissue and attains lethal quantities long before coagulation of protein occurs. Death of cells from high temperature results when there is an irreparable destruction of the molecular structure of the cytoplasmic body.

The dwarfing effect of high temperatures may be seen in the growth of our common garden asters when planted in the South. In regions where the summer temperatures average 95 to 100°F., they may produce an unbranched stem 6 or 10 inches high with a single flower not over 1 inch in diameter, as contrasted with the thrifty, branched growth with numerous chrysanthemumlike flowers that are normal in the more temperate climates of the North.

The writer is inclined to attribute certain failures of summer-seeded wheat that have been observed in the Pacific Northwest to long days combined with abnormally high temperatures. It has been observed that, under certain conditions, winter wheat that was seeded about July 15 to Aug. 1 either made a very poor growth and gave a reduced yield or failed almost entirely. Entire fields of such early-seeded wheat have been noted when the plants that did come through the winter made a sickly, chlorotic, stunted growth and produced few or no heads. This permanent stunting apparently results when the young seedlings are subjected to temperatures which fluctuate slightly above or below the maximum temperature for growth.

During the periods of intense heat in the summer in the northern sections or frequently in the hot season in southern latitudes, there may be more or less burning of leaves, flowers or fruits. With leaves, there may be a death of marginal tissue, or internal necrotic areas may be formed, in many cases the effects resembling drought injury. Injuries of this type may result when the tissues are suffused with water and the atmosphere is humid, or very similar effects may follow when transpiration is greatly accelerated by low humidity and high temperatures. The tipburn of the potato is characterized at first by a slight wilting and yellowing of the tissues at the extreme tips or sometimes at the margins of potato leaflets, followed later by a browning and death of the tissues, until in the extreme cases the entire leaflets are involved. It has been

pointed out that the principal factors operative in producing this disease are *heat* combined with intense light.   The trouble appears to reach its greatest severity on the crest of heat waves or high-temperature periods, and it is significant that such periods are generally marked by minimum humidity and very intense sunshine.   This physiological tipburn is distinct from "hopper burn" caused by the feeding of the leaf hopper, *Empoasca mali*.

The appearance of sunscald spots on the foliage of plants grown under glass is not uncommon.   Isolated spots, lines of spots or dead streaks

Fig. 221.—Typical tipburn injuries.   Leaflets *A, B* and *C* were more or less rolled; leaflets marked *D* were not rolled to any extent.   (*After Lutman, Vt. Bul.* **314.**)

may be formed.   These are attributed, in a large part, to the concentration of the heat by bubbles in the glass which act as burning lenses, and the lines of spots or streaks result from the shifting position of the sun. It has also been noted that water drops may act in a similar fashion as burning lenses, both in greenhouse cultures and in plants grown in the open.   Spraying of delicate plant structures during the heat of the day may promote injury for the above reason and also from increased sensitiveness to heat due to a modified water relation.   Heat cankers may result from high summer temperatures when trees have their trunks or limbs exposed as a result of clearings or cutting for roads or from any removal of shade, which has served as a protection.   Bark cankers of this origin should not be confused with the so-called "winter sunscald."

The scalding of flowers frequently results from high temperatures. The browning of the rays of dahlia flowers in the early blossoms produced during the periods of high temperature and intense sunshine is a notable example.   Perfect flowers are formed later in the season when lower

temperatures prevail. Many fruits suffer from sunscald or sunburn, especially those which are succulent. Even fruits like the apple may be severely burned, during periods of intense summer heat, especially the sun-exposed face of fruits hanging on the outer branches. Injuries of this type may be intensified by lime-sulphur or other sulphur spray (see Spray Injury) but may occur independent of any spraying operations. Strawberries, grapes and other fruits may frequently be scalded when they are exposed to intense sunlight and high temperature following a humid period in which absorption was active but transpiration checked. Sunscald injuries are due not to the direct action of the air temperatures but to "excessive heat generated in exposed parts of plants by sunlight absorption."

Fig. 222.—Heat canker of pine and beech seedlings. (*Redrawn after Münch.*)

The sunscald of tomatoes is also a very common cause of complaint. Heat conditions which are not sufficiently intense to produce localized injury may accelerate the ripening processes and lower the keeping qualities of the fruits that are prematurely ripened.

Heat defoliation may occur in both deciduous and evergreen trees, and, according to various observers, the more exposed leaves, or those occupying a peripheral position, are less likely to be cast than those in a more protected position within the crown. Although leaves can reduce their temperature by an active transpiration, under certain conditions their temperature may rise above the maximum which their tissues can tolerate, and heat casting is the result. It is the belief that the inner leaves suffer first because of their greater sensitiveness to heat and also because the radiation of heat from them is retarded by the more exposed foliage. Seedlings of coniferous or deciduous trees or of herbaceous species may be seriously injured or even killed by high temperature, the injury being localized in the stem just above the ground level, thus resembling damping-off due to fungi. This trouble has been called "white spot," especially on conifers. The order of heat tolerance for four evergreen seedlings was as follows: lodgepole pine, yellow pine, spruce and Douglas fir. It was suggested that temperature may be the critical factor which limits or prevents natural reproduction. Temperatures of the surface soil in nurseries have been shown to range from 53 to 54°C. in ordinary soil, and even higher in sand soils (54 to 72°C.). Under artificial conditions, resistance of evergreen seedlings increases with age and size or

mass, based on stock one to four years old, and are more tolerant in dry air (15 per cent) than in moist air (85 per cent). Similar heat injury has been described for cowpeas, rye, buckwheat, beans, oaks, maple and vetch. Severe injury to cucumber seedlings resembling damping-off was observed by the writer when heat was the only factor that seemed responsible. It seems probable that heat injury to young tender seedlings is much more common than recorded experiences would indicate. Severe injury of this type to a field crop is well illustrated by the heat canker of flax (see Special Treatment).

Not only seedlings but mature herbaceous plants may succumb from high temperatures during the intense heat of southern summers. A single illustration will emphasize this behavior. The common garden nasturtium will thrive and flower luxuriantly in the early part of the growing season in middle Texas but suffers from "sunstroke" during the midsummer period of cloudless skies and temperatures ranging from 95 to 100°F. or more.

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LOW TEMPERATURES

Living plants, either growing or dormant, or plant products are likely to be subjected to temperatures sufficiently low to cause either death or injury. The character and extent of the injury will vary with the temperature and the condition of the plant or plant structures.

General Effects of Low Temperatures.—In discussing the general effect of temperature upon growth, it was pointed out that for each species, variety or strain there is a certain degree of warmth, the *optimum* at which growth is most rapid. As the temperature sinks below this optimum, growth becomes less and less rapid and finally ceases at the *minimum.* This retardation or checking of growth is the inevitable result of low temperature. A second effect of low temperature is the prevention of chlorophyll formation or the slower construction of this pigment, with the result that parts normally green may become yellow. In some plants or plant parts, cold causes the development of red pigment, which apparently obscures the lesser degree of chlorophyll development. When the temperature sinks to a sufficient degree, freezing of plant tissue results, and death may follow, or, with the rise of temperature, the frozen tissues may thaw out without any appreciable injury.

The final result following exposure to low temperature will be variable, depending upon specific peculiarities of plants, moisture relations, length of exposure, degree of cold and other internal or external factors. It may suffice to say that the final results in the action of cold will fall into three groups: (1) recovery or return to normal with the advent of temperatures

favorable to growth; (2) either the parts may be lost, malformed or disfigured or, in annuals, the entire plant may become a permanently deformed cripple for the rest of the growing season or, in perennials, may lead a struggling existence in a condition of lowered vitality for a period and finally attain normality, or the derangement may become more pronounced and end in death; and (3) the injury may be of the *acute* type in which sudden death of the plant is the outcome.

How Freezing Causes Injury.—There has been much discussion as to how freezing causes the death of plant cells. The early theory of cell rupture due to expansion of the cell sap was soon discarded, because it was shown that water was withdrawn from the cells into the intercellular spaces where the freezing occurred. It is now generally agreed, however, that this withdrawal of the water from cells is the serious feature in freezing injury. Several different theories for the injury have been offered:

1. *Cold Death by Poisoning.*—The withdrawal of water is thought to concentrate the cell sap so as to leave substances actually toxic to the protoplasm.

2. *Mechanical Injury.*—With this concept, death results from "mechanical injury of the protoplasm caused by the compression of the ice crystals which accumulate in the intercellular spaces."

3. *Destruction of Protoplasmic Structure or Architecture.*—The loss of the semipermeable character of the protoplasm permits the water to pass into the intercellular spaces. If this structure is permanently lost, death results, and the intercellular moisture which cannot be reabsorbed is soon lost by evaporation. It has been stated that protoplasmic changes and the consequent death of the cell result "from the formation in the protoplasm of relatively large ice crystals and the consequent alterations of the space relations of the phases constituting the colloidal complex of the protoplasm." Recovery following freezing would take place only if thawing permitted the regaining of the original space relations.

Variation in Cold Resistance or Hardiness.—Plants show wide variation in their tolerance of low temperatures. It is well known that tomatoes, potatoes, beans, cucurbits, corn, dahlias, etc., are extremely frost sensitive, generally suffering acute injury with the first formation of ice crystals. Other annual plants, such as spinach, lettuce, the various cereals, etc., are much more frost resistant, and in certain regions hardy varieties may even behave as winter annuals, beginning their growth in the fall of one season and completing it during the next. Contrast, for example, the difference in the frost resistance of dahlia roots or potato tubers and the bulbs of such hardy plants as tulips, narcissuses, jonquils, etc. In general, the crops of our temperate regions which have developed the biennial habit, such as carrots, beets, parsnips,

salsify, cabbage, etc., are frost resistant. Some frost-sensitive plants which behave as annuals in temperate regions may become perennial in the moderate climate of southern regions. The perennials exhibit all gradations from tender to extremes of winter hardiness, and one of the tasks of the plant breeder is the production of hardy varieties of farm crops, fruit plants and other desirable plants which will extend the range of profitable production.

The Basis of Hardiness.—Many different theories have been offered to explain hardiness or resistance to injury from freezing. The most probable are based on the Müller-Thurgau theory of death by water loss incident to the destruction of the protoplasmic structure. Since water retention is the basis of hardiness, the main factors which are concerned in water retention may be noted: (1) the property of the hydrophylic colloids of the cell to hold water by such phenomena as adsorption, adhesion and molecular capillarity; (2) the carbohydrate content, since many hardy plants show a higher carbohydrate or sugar content than tender varieties; and (3) the osmotic concentration of the cell sap of the vacuoles owing to various solutes or soluble compounds. Neither (2) nor (3) is a constant accompaniment of resistance to freezing. With the recognition of the part played by hydrophylic colloids, studies have been directed to the determination of the influence of specific colloids.

Attempts have been made to correlate hardiness with other measurable factors, that is, quantity of press juice, moisture content, total solids, amino acids, organic nitrogen or viscosity, but none has proved to be an infallible indicator. The only recognized reliable test of hardiness is artificial freezing at controlled temperatures with the determination of injury.

FROST INJURY

This discussion will include injuries which result from low temperatures after plants have started into growth in the spring and during the period of vegetative activity before they have matured and entered their period of winter dormancy.

Low-temperature Injuries to Leaves and Young Shoots.—The effects vary with the degree of cold and the sensitiveness of the individual or the species and include: chlorotic bands or spots or general chlorosis or yellowing; the formation of a red pigment, anthocyanin, in organs normally green; blistering, crinkling, curling, puckering, irregular laceration or shot holing of leaf blades, sometimes with reduction in size; and the partial or complete necrosis or blighting of leaves and vegetative shoots. Severely injured leaves appear water-soaked at first, owing to the formation of ice crystals in the intercellular spaces but, with the melting of the ice and loss of water, remain limp and flaccid, and become rapidly discolored, dry out and soon fall or weather away.

Injury to Blossoms and Young Fruit.—Blossom buds or blossoms may blight and drop without setting fruit, or later frosts may affect fruit after it has already set and cause shedding, reduction in size, malformation,

FIG. 223.—Frost curling of apple leaves.

FIG. 224.—Frost-lacerated and deformed leaves. *A*, horse chestnut; *B*, maple; *C*, lilac; *D*, apple; *E*, linden.

internal necrosis or sterility. The essential organs of the flower, stamens and pistil are generally more sensitive than the accessory parts. Injury by slight freezing may be marked by a discoloration of the pistil, which

becomes brown or black, while the surrounding parts may remain nearly normal. The killing temperatures for fruit plants vary for the different species and varieties: (1) for closed buds that are showing color, 20 to 30°F.; (2) for open blossoms, 25 to 30°F.; and (3) for setting fruit, 27 to 32°F. The "black eyes" of strawberry blossoms which have suffered a knockout blow by Jack Frost are typical of frost injury. Complete or partial sterility of wheat heads may result from low temperatures about

A   B

Fig. 225.—Diagrams of sections of normal and frost-injured apples. *A*, soon after the petals have fallen, showing central necrosis; *B*, when ¾ to 1 inch in diameter, showing peripheral necrosis.

the time of emergence from the boot, and lodging or breaking over may result from the killing of the meristematic tissue at the base of internodes.

Frost Russeting of Orchard Fruits.—The skin of fruits normally smooth may show brown, rough areas, or *russeting*. The russeting may

Fig. 226.—One normal and several frost-injured heads of club wheat. (*Photograph by B. F. Dana.*)

be more or less diffuse or scattered, localized at either stem or calyx end, confined to an equatorial band producing the so-called "belted or banded" fruits, or more rarely in the form of russeted knobs or even russet rings, ½ inch or slightly more in diameter.

Frost Injury of Sensitive Annuals.—It is a matter of common observation that frost-sensitive annuals suffer acute injury from spring freezing,

but it is not so generally understood that less severe injury may cause permanently dwarfed and crippled individuals. Such injured plants may make a poor growth and survive for the entire growing season, with little or no fruit production. This behavior is not uncommon in frost-sensitive plants like beans or cucumbers.

Annualism in Biennials.—Plants like carrots, beets, parsnips, etc., normally make their vegetative development with the storage of reserve food during the first season and blossom and fruit the second season.

Fig. 227.—"Belted fruits." *A*, apple; *B*, pear.

Field experience and experimental data have shown that spring frosts during the younger stages of growth may furnish the stimulus to initiate the reproductive function. The frosts may cause a cessation of growth, and this has the same effect as a period of winter rest, with flowering more likely if the frost is late in the season. Other nutritive disturbances than those caused by frost may lead to annualism.

The Prevention of Frost Damage.—Three general principles used in the protection of growing plants from frost are: (1) conserving heat; (2) mixing or stirring of the air; and (3) adding heat.

Heat may be conserved by covering the ground or plants with glass, cloth or lath screens, paper caps, etc., by surface sanding of muck soils, by flooding as in the case of cranberry marshes or muck crops and by overhead irrigation for some truck crops. Smudge fires of damp straw or manure are sometimes used to form a protecting blanket of smoke. Chemical smoke screens, fogs of moisture particles or chemical-bearing

fogs have been suggested. Several devices for stirring the air with large power-driven fans have been used with limited success; also by a low-flying airplane over potato fields in peat soil. Adding heat is accom·plished by lighting a large number of small fires appropriately placed throughout the area to be protected. Various types of orchard heaters for use with either oil or solid fuel are available and have been extensively used with very satisfactory results. By the use of heaters, the temperature may be raised sufficiently (6 to 10°) to afford safety except in unusual drops. Potash fertilizers have been reported to give increased frost resistance to certain crops.

Fig. 228.—Net and ring types of frost necrosis of potato. *A*, intense net discolorations in both medulla and cortex; *B*, intense ring type somewhat complicated by blotch. (*After Jones, Miller and Bailey, Wis. Res. Bul.* **46.**)

Cold Injury to Harvested Crops.—Low temperatures at the end of the growing season or during the period of dormancy or storage may cause injury to root crops or fruit. The principal effects of exposure to low temperatures are: (1) changes in composition, flavor, color or texture; (2) susceptibility to injury from handling while frozen; (3) premature breakdown; (4) localized internal or external killing or a *frost necrosis;* (5) increased susceptibility to decay; and (6) freezing solid, after which the tissues become soft, and watery and discolored and disorganized with the rise in temperature.

Aside from the extreme injury of freezing solid, potatoes may turn sweet when stored for a number of weeks at temperatures close to the freezing point. Tubers exposed to low temperatures, but not sufficiently long or low to freeze solid, may develop internal discolorations. ·Three different types of internal frost necroses have been recognized: (1) the *blotch type,* irregular patches mostly in the cortex and ranging in color

from a slight metallic tinge to almost sooty black; (2) the *ring type*, or necrotic areas in or adjacent to the vascular ring; and (3) the *net type*, evident as browning or blackening of the fine ramifications of the vascular elements.

Fig. 229.—Blotch type of frost necrosis of potato. *A*, longitudinal section; *B*, cross section showing intense vascular and cortical blotches that were evident on the exterior as dark areas. (*After Jones, Miller and Bailey, Wis. Res. Bul.* **46.**)

Turning sweet of potatoes is noticeable at 35°F., but the transformation of some of the storage starch into sugar is more rapid at 32°F. Internal necroses result from temperatures between 32°F. and the freezing point of potato tissue, which is around 26 to 28°F. or slightly higher. The injury depends on the degree of cold and the length of the exposure, and it will vary with the variety or lot.

Special studies have been made of freezing injuries of apples, tomatoes, grapes and other fruits. The freezing point for apples has been shown to vary from 26.87 to 30.16°F. with 28.5°F. as an estimated average; average of 30.46°F. for tomatoes; and a range from 23.63 to 28.66°F. for different varieties of grapes. Root and fruit crops may suffer injuries from low temperature as a result of delayed digging or harvesting, from too low temperatures in pits, storage cellars, in cold-storage warehouses or in transit to market.

WINTER INJURY

This phase of low temperature effects includes injuries which result from low temperatures to plants after the end of the growing season and before growth starts in the spring.

Types of Winter Injury.—The degree of winter injury to perennial plants, either herbaceous or woody, varies from the most acute effects resulting in death of the plant to localized injury affecting certain organs and evident by either internal or external changes.

The winterkilling of winter annuals like wheat, spinach, onions, etc., is frequently responsible for heavy losses. In some localities which normally produce winter wheat, 75 per cent of the fields may be killed out to such a degree as to necessitate reseeding. The death of herbaceous perennials such as alfalfa or strawberries during unfavorable winters is of common occurrence. Injured plants may be killed outright, while in other cases, with the advent of spring, there is still a faint spark of life and growth starts, but the vitality has been so weakened that death follows this last struggle for survival. Trees and shrubs, including fruit, shade and forest plantings, frequently suffer severely during test winters, even in regions to which they are supposed to be adapted. When woody plants are apparently normal at the close of the growing season, but fail to start into growth in the spring, the presumption is in favor of death by freezing.

Twig Blight or Dieback.—Dieback is a normal phenomenon in species having an indefinite or undeterminate annual growth, such as rose, sumac, elder, brambles, etc., but, in species with a definite or determinate annual growth, the twig completes its elongation and develops its terminal and other buds by the end of the growing season. When twig blight is general in such species or when the death of larger branches takes place, serious injury may result to the life of the tree and to its productive capacity. Species of trees such as cherry and other stone fruits which show a natural tendency to gummosis when tissues are killed will generally show conspicuous gumming of winterkilled branches. Dieback is not confined to deciduous trees, but may cause severe injury in forest and cultivated evergreens. Sometimes deciduous species may have the leaves "frozen on" before the abscission layers are formed, or evergreens may show an abnormal shedding of needles.

Bud Injury.—Injury to buds, especially flower buds, may occur independent of dieback or in connection with it. In severe injury, the internal tissues will show a brown coloration, and the injured bud may dry up and drop off in early spring. Such injury is common in peach and other stone fruits but less common in apple and pear. In the latter, severe temperatures may cause a complete kill, but lesser injuries may occur leading to the formation of flowers with malformed parts, followed by the production of malformed fruits. Some of the effects are dwarfed, flattened, tomato-shaped, seedless or coreless types. Bud injury is likely from early December freezes or from late freezes following unreasonably warm weather which starts the buds into activity.

Root Killing.—Under severe conditions, localized or general root killing may result. If there is a general dieback of young roots, the absorbing power of the root system may be seriously impaired, with the result that the trees make a poor growth. A more general killing of the large roots may cause wilting and death of branches or of the entire tree, even some time after growth has started in the spring.

Frost Cankers.—Localized killing of the bark or of the bark and cambium may occur on the exposed faces of large limbs, at the crotches,

Fig. 230.—Section of tree trunk showing both longitudinal and radial frost cracks. (*After Neger.*)

upon the southwest side of the trunk or at the base of the trunk. The killed areas may be small or extensive, superficial or extending down to and including the cambium, and on one surface or completely girdling the branch or trunk. Winter sunscald cankers and collar rot seriously menace the life of trees (see Special Treatment).

Frost Cracks.—Winter temperatures cause two types of splitting of the trunks or large limbs of trees: (1) longitudinal cracks which extend radially from the bark through the sapwood to the center of the trunk or beyond; and (2) "cup shake," or cleavage along an annual ring, involving a small or an extended part of the circumference.

The Little-leaf Condition.—Under certain conditions fruit trees, especially apples, show a trouble which has sometimes been called the "little-leaf disease." This is distinct from the so-called "rosette" and appears to be a type of winter injury. In the most severe type, the leaf buds burst the bud scales and expose the young leaves which may soon wither and

dry up without any further growth, or, in the other extreme, wilting and death may be delayed until the leaves are half size or full size or even until later in the season.   Between these two extremes, different degrees of injury may be found.

Blackheart or Internal Necrosis.—The pith and heartwood, or the sapwood also, may show a pronounced blackening following severe winter conditions.   Blackheart is very common in apple trees in the northern ᵣange of apple culture, and such trees may continue to grow for years with little or no evidence of the internal derangement, but they appear to be more susceptible to the inroads of wood-destroying fungi.   Silvering of the foliage appears to be an aftereffect of this type of injury, but both blackheart and silvering also result from invasions by the silver-leaf fungus, *Stereum purpureum.*

Factors Affecting Winter Injury.—The degree and the type of winter injury will be influenced by the condition of the plant as well as by the combination of unfavorable weather conditions.   It is not alone the absolute temperature or the minimum cold which is of importance but the time in the period of dormancy when the cold is experienced and whether gradual or sudden changes occur.   Periods of zero weather in December are very likely to cause severe injury, because the tissues have not yet
· become hardened by the longer action of moderate cold.   Periods of zero weather following moderate weather which starts tissues into activity are also most likely to induce heavy injury.   The presence or the absence of a snow cover at the time of the heavy freezes is also a matter of much importance.

The type and the severity of winter injury will be influenced by the following factors: (1) the species or kind of crop and the variety within the species; (2) the age in the case of perennial plants; (3) the degree of dormancy of the plant as a whole, or of special parts at the time of critical temperatures.   Some of the factors which affect not only dormancy but other features which influence hardiness are: (1) kind or degree of pruning and time of pruning; (2) the amount of crop produced the previous season; (3) the heat and light income during the growing season, especially during the late fall; (4) the physical characters of the soil and subsoil; (5) the natural fertility of the soil or the fertilizing practices; (6) the site with reference to soil moisture or drainage conditions; (7) the time of irrigation and the amount of water used; (8) the presence or absence of cover crops; (9) cultural practices, for example, time of seeding or furrow seeding for cereals.

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UNFAVORABLE LIGHT RELATIONS

Before considering the ways in which disturbances of the light relation may bring about derangements in the life of our cultivated plants, brief record should be made of the part which light plays in some of the important physiological processes.

The Function of Light in the Life of the Plant.—Light is essential (with few exceptions) to the formation of the green pigment, chlorophyll, and furnishes the power or energy by which the chlorophyll apparatus is able to use the crude materials, carbon dioxide and water in photosynthesis, or the construction of carbohydrate food. The output of our plant factories depends upon the intensity, duration and quality of light; hence it must be evident that the amount of plastic material available in plants for assimilative and growth processes bears a definite relation to illumination.

That light has a direct effect upon living protoplasm is evidenced by various heliotropic curvatures or movements of plant organs, which direct them either toward or away from the light, by heliotactic movements of motile plant protoplasts or by intracellular changes with variations in the intensity of light. Since protoplasm exhibits a marked sensitiveness to light, it is only reasonable to expect that growth will be affected by light conditions. Growth is favored by diminished light intensity and retarded by bright illumination. During the daylight period, the energy of the plant is directed to the work of food manufacture; while, during the night period, photosynthesis ceases, and the reserve of plastic materials which were accumulated is available for constructive work. Ordinary daylight does not cause a cessation of growth but merely a retardation. This behavior is illustrated by the growth increments of certain organs which in darkness may be more than double those for similar periods of ordinary daylight. While light is not essential to the germination of most seeds, it is the stimulus that in some cases unlocks the chemical changes that initiate germination.

Transpiration is a third physiological process affected by light. The effect may be either direct upon the living substances or upon the cell machinery or indirect by modifications of the plant structures. Since

transpiration is not comparable to simple evaporation from an exposed water surface but is a process controlled more or less by the chemical and physical properties of the protoplasmic body from which the moisture must be withdrawn, light may affect transpiration by modifying the permeability of the protoplasm and thus accelerate or retard the process. By affecting the osmotic pressure in the guard cells or adjacent epidermal cells, light may cause an opening or closing of the stomata and thus promote or retard *stomatal transpiration*, while cuticular transpiration will be influenced by the thickness of the epidermal walls, their cutinization, the character of the cuticle and other structural features which are modified by light intensity.

Shade Plants and Sun Plants.—Brief reference may be made to the fact that some plants in their natural habitats may be able to make their best growth only when exposed to the full intensity of normal light, while others thrive best in partially shaded localities in which the light is of moderate intensity. Thus we may have sun plants at one extreme and shade-loving plants at the other, while others are less sensitive to their light environment. These natural peculiarities must be taken into consideration in providing favorable conditions for plants under cultivation. Shade-loving plants may suffer injury if exposed to the full intensity of the sun, or sun plants may make a poor growth if planted in poorly lighted environments. Because of the injurious effect of intense light, lath screens giving approximately half light are used for certain shade-loving crops like ginseng or golden seal, which normally grow in forest areas, and also for the seedlings of conifers and some other forest trees.

General Effect of Light Deficiency.—For a normal thrifty development, a certain intensity and duration of light are essential. It is difficult to fix upon any definite optimum, but it must be recognized that, when the light income of a plant sinks below the minimum requirement or rises above a maximum, the plant will cease to thrive. The *optimal light income* denotes the amount of light (measured by intensity and duration) which will induce the best growth or produce a type of development that is most nearly normal. As the light income drops from the minimum to zero or complete darkness, the plant may undergo gradual, formative or structural changes, including alteration of color and peculiarities of structure which are characteristic of the condition known as etiolation. The sickly yellow plant that has been entirely deprived of light represents the extreme of etiolation, while, with exposure to light, the symptoms become less and less evident until normality is attained.

The changes which are characteristic of etiolation are as follows: (1) The *abnormal elongation* of stems (internodes) and petioles or of leaves that are normally elongated and have a basal or intercalary meristem, thus producing shoots that may be characterized as "spindling." This

modification occurs in plants with stems that are normally elongated and also in those of the rosette habit. (2) The pronounced reduction in the size of the leaves (shade leaves in many cases are larger than sun leaves). This feature is characteristic of dicotyledons, while monocotyledons generally show an elongation and narrowing of the leaves, although deviations from this behavior may be found in both groups. (3) The reduction in the amount of chlorophyll or its complete disappearance (in darkness) and consequently the slowing up of photosynthesis or a complete cessation of food manufacture. In complete etiolation, growth can continue only at the expense of food reserves of seeds, modified stems or other storage organs. It should be noted here that etiolation is not the result of a checked or inhibited photosynthesis, since plants will not make a normal development in darkness even when supplied with an abundant reserve of plastic food. (4) The suppression of the reproductive function, as illustrated by sterility, lessened flower formation or the complete absence of blossoms as a result of shortage of food. Plants like hyacinths, tulips and narcissuses, with an abundant food reserve, will develop nearly normal blossoms in complete darkness. (5) A soft or succulent type of growth. The stems are more slender and leaves thinner, cell walls are more delicate, and there is a poorer development of mechanical tissue. The general effect of diminished light is to cause a poor development of the palisade parenchyma of leaves, so that shade leaves may not be more than half as thick as those developed under normal illumination.

Plants grown under poor light conditions wilt more readily than normal plants when exposed to bright light. This may be illustrated by the behavior of cucumbers grown under glass in northern latitudes during the cloudy and short days of late fall and early winter. The leaves have a poor color, slender, elongated petioles and poorly developed mechanical tissue. When subjected to the bright sun the plants may wilt and suffer injury depending on the suddenness and duration of the changed light relations. This is only an extreme illustration of the effect of poor illumination, and undoubtedly many minor injuries pass unnoticed.

Poor light, or "partial etiolation," renders plants more susceptible to the attacks of fungous diseases. Undoubtedly the host modifications play an important part in this increased susceptibility, while, at the same time, the reduced light intensity offers conditions more favorable to the growth of fungi. The light factor is not the sole favoring condition, for diminished intensity of light is generally accompanied by increased humidity of air, which may also affect both host and parasite. As an illustration, it may be noted that lettuce grown under glass sometimes suffers severely from leaf blight and stem rot, due to Botrytis, during the

dark, cloudy days of winter, while the trouble may largely disappear with the advent of days of continuous sunshine. Lack of light together with excessive moisture increases the susceptibility of foliage to injury from fumigants, for example, burning from hydrocyanic acid gas, while insufficient light income has an important bearing upon one type of spray injury (see Lime-sulphur Injury). Insufficient light produces a type of growth in which maturity is delayed, and, consequently, winter injury is more likely. It seems that winter injury in some environments is accentuated not alone by the moderate temperatures of the fall preceding freezing weather but also by the lowered light income due to cloudy, foggy weather.

Etiolation in Horticultural Practice.—While etiolation represents a derangement of normal physiological processes, it may be utilized to produce desirable modifications of behavior or to produce structural modifications which render the plant more desirable as a commercial article. Hyacinth bulbs planted in flower pots in the fall sometimes show a delayed development of the leaves and the flower stalk, which remain short. This may be prevented, in part at least, by covering the bulb and bud with a cap of dark paper, which will exclude the light. This induces etiolation, and, as a result, the leaves and flower stalk elongate. Similar use may be made of withdrawal of light to induce the formation of blossom shoots in other plants.

Etiolation or blanching gives a desirable quality sought in certain vegetables, for example, asparagus, endive, celery, French globe artichoke, head lettuce and cabbage. The etiolation may be induced by depriving the parts to be blanched of light; but in numerous cases, cultivation and selection have produced varieties that are partially self-blanching, the failure to green becoming a hereditary character. Etiolation improves or modifies the flavor and produces crisp, juicy and tender tissues, a desirable feature, especially in salad plants.

General Effect of Intense Light.—If plants are in danger of sunstroke, they are not able to flee to a shaded or secluded spot where the light is less intense, but they do, by certain habits or by active responses, show a sensitiveness to the intense light of the environment in which they live, and these may be purposeful responses which serve as a protection against injury from intense light. A number of illustrations may be noted. The chlorophyll apparatus of the leaf is in the most favorable position for receiving the full intensity of the light when the rays fall most nearly perpendicular to the leaf surface and would be best protected from injurious effects of too intense light if the rays are parallel to the leaf surface. This protection is attained in some sun plants by the erect or ascending position of the leaves or by their orientation in such a way that their surfaces face either east or west, as in so-called "compass plants." In

other cases, as in many legumes, the leaflets, which in moderate light are spread out to receive the full-light income, fold either upward or downward in pairs when the light becomes intense (during midday in the summer) and thus place the leaf surfaces parallel to the incident rays of light.

In many cases in which protection of the chlorophyll apparatus cannot be obtained by the position of the leaves, the chlorophyll bodies change position within the leaf cells. During moderate illumination there is a tendency for them to be massed or grouped on the surface faces of the cells, while with too intense illumination they are shifted and arranged along the walls vertical to the surface. This change of position may cause a deep-green color in the shade and a less intense or paler color in the intense light and will explain the so-called "shadow pictures" of shading organs upon brightly illuminated leaf surfaces.

With increase in the intensity of light, photosynthesis will increase up to a certain point, and then, with further increase in intensity of light, the photosynthetic activities remain about constant, but for a short time only. If the intensity becomes too great or the optimum is exceeded for too long a period, the construction of carbohydrate food becomes less and less active and may finally be checked entirely. During the exposure of green plants to ordinary illumination, the green pigment, chlorophyll, is being constantly oxidized, but it is being constantly reconstructed, hence the change is not evident. Under conditions of intense illumination, light-sensitive plants develop a pale or yellowish-green cast or even a bronzing of the leaves. Under such conditions, the chlorophyll is oxidized somewhat faster than it is reconstructed. This behavior is frequently noticed when plants that are normally shade-loving, for example, those that select the shaded forest as their habitat, are suddenly moved from glasshouses with poor illumination to the bright sunlight of the open. The sensitiveness to intense light often varies with the age of the plant, for example, the seedlings of many trees are not able to withstand the direct sunlight, while older trees suffer no derangements from exposure to the same light intensity. In the more sensitive species, continued exposure to intense light may kill the protoplasm of cells in leaves, stems or fruits, and browning, burning or blighting of localized spots or more extended areas may be the final result. Specific sensitiveness to light and the conditions of moisture, temperature and light which have prevailed previous to the exposure to intense light have an important bearing on the type and degree of injury which results (see Unfavorable Water Relations, page 478; also High-temperature Injuries, page 505).

A spotting and streaking of beans in Colorado, at relatively high altitudes, affecting pods and foliage has been shown to be due to the action of light rather than heat. Under the conditions prevailing at high

altitude and with low humidity, the light reaching the growing plants contained a high proportion of ultraviolet rays, and it was the conclusion that the sunscald was caused by these rays of short wave length. Observations indicate that injury of this type will be more severe at high altitudes than at low altitudes. A sunscald of soybeans and cowpeas, as it occurs in Arizona, appears to be very similar to the sunscald of beans in Colorado.

Photoperiodism.—The response of plants to light depends on three factors: (1) the intensity of the light; (2) the wave length or quality; and (3) the duration of the light action. The earlier work was devoted primarily to studying the effect of the first two of these factors, while it is only since the researches of Garner and Allard (1920) that the significance of the daily period of light in the growth and reproduction of plants has been appreciated. Since that time numerous investigators have contributed to our knowledge of the subject. A discussion of recent contributions is presented in some detail by Garner (1937) and by Whyte and Oljhovikov (1939).

The length of daylight to which a plant is exposed is expressed in the type of growth that results. In nature, the establishment of a species in a given environment depends on its ability to flower and produce seeds, and this is possible only when the favorable length of day is presented combined with proper temperatures. For example, soybeans do not give the expected reaction to day length when grown at high temperatures. Many other species have been shown experimentally to give an altered reaction to length of day when grown at different temperatures. The favorable length of day for a plant is termed its *photoperiod*, and *photoperiodism* is the response of a plant to the relative length of day and night. Most of the studies on photoperiodism have been concerned with herbaceous annuals or biennials, but similar responses have been shown for a number of woody species, and it has also been shown that the photoperiod affects the tuberization. For example, species that produced good tubers in equatorial South America formed none when grown at Leningrad.

In the nature of their response to length of day, two extreme types of plants may be recognized: (1) *short-day plants*, or those which tend to a vegetative development with increase in stature when exposed to long daily periods of light and flower and fruit only when the light period or the length of day is suitably decreased; and (2) *long-day plants*, or those which show an altered development and fail to flower when subjected to short days but readily form flowers under the influence of suitably lengthened periods of daylight. In addition two other groups have been recognized: (1) *intermediate*, including certain species or varieties that flower if the day is either sufficiently short or sufficiently lengthened; and (2) *indeter-*

minate, in which flowering is not related to day length. The length of day is a factor of importance affecting the natural distribution of plants in different parts of the world.

The plasticity of plants under the influence of variation in the length of day and night to which they are exposed is very marked but finds its most striking expression in the effect upon flowering and fruiting, while various chemical and structural changes may be accompaniments. Flowering and fruiting may be retarded or accelerated, the type of vegetative growth modified to lead to gigantism or to dwarfing, while the laying down of reserve foods in bulbs, tubers or roots may be inhibited or seriously impaired. The anatomy of leaves or other plant organs may be changed, and the production of fiber may be affected, while the ratio of flowers of different sex may be modified (corn) and in some cases even a reversal of sex may result (hemp).

In the cultivation of plants, the desired end may be production of a luxuriant vegetative growth, or *gigantism,* with an inhibition or delay of flowering or fruiting, although in a large percentage of our ornamental or crop plants the production of flowers or of fruit is the feature of commercial value. It is not possible within the space available to give any detailed discussion of the numerous illustrations of photoperiodism which have been studied. Practical use has been made in greenhouse cultures of increasing the light period with electric light and of reducing it by covering with black cloth for a part of the day.

Plants in their native haunts have become adjusted to the specific light periodicity of their environment, but in our agricultural and horticultural practice they may be subjected to light periods to which they are not suited: (1) by field planting in regions with too short or too long days; (2) by date of seeding in the field so that the optimum light periods are not experienced during the growing season; and (3) by cultures under glass which are independent of the season as far as temperature and moisture are concerned, but with seasonal variations in the length of day. Photoperiodism of plants has an important bearing upon the success of cultures under artificial illumination and emphasizes the fact that the desired results can be attained only with careful attention to the varying light requirements of different varieties. Failures to attain the desired ends in farm or horticultural practice may be due to a lack of acclimatization of our cultivated species with respect to the light relation or to our failure to understand the specific light requirement of the variety.

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APPLE SCALD

Apple scald occurred to a limited extent before the days of cold storage, but it has been much more in evidence since the extensive refrigeration of fruit during storage or in transit to market. The disease was well characterized as early as 1896, but its real cause was not discovered and a control perfected until the work of Brooks, Cooley and Fisher in 1917 and later.

Symptoms and Effects.—Scald is characterized by the discoloration of the skin of stored fruit, the color varying from a faint-brown tint in mild cases to a pronounced brown involving the entire thickness of the skin in more severe cases. The scald appears first on the lighter surface of the fruit, where it is most severe and spreads until all or a large part of the surface may be involved, depending somewhat upon the maturity of the fruit at the time of storage. Green or undercolored skin is the most susceptible, yellow of medium susceptibility, while deep red is generally the most resistant.

Severe scald may be followed by physiological decay or internal breakdown, which may involve much of the interior. The scalded areas are soon invaded by rot-producing fungi which find the dead tissues an easy avenue of entrance and thus complete the work of destruction.

The symptoms described above have sometimes been designated as "common," "superficial" or "hard scald" to distinguish them from another trouble of somewhat different origin known as "soft" or "deep scald." In both common scald and soft scald, the injured tissues may be invaded by Cladosporium or other fungi and become spotted with black.

Scald may appear in common or home-stored fruit, but it is of most concern as a disease of commercial storage or of market fruit. It may develop on fruit while still in commercial storage, but it makes its most rapid development after the apples have been moved to the warmer temperatures of the market or the home. Apples may appear to be in perfect condition upon removal from cold storage but become so scalded a few days later that their market value may be reduced 15 to 30 per cent or more.

Before the days of present effective control, market inspection reports showed that scald caused almost as much loss on the market as blue mold. This did not take into account latent scald that appears later; hence the

Fig. 231.—Common scald of apple.   (*After Brooks and Fisher.*)

total losses were probably higher than from any other market trouble. Apple scald caused losses or handicaps to the apple industry in a number of ways: (1) by depreciation in price owing to actual occurrence of the

Fig. 232.—Soft scald of Jonathan apples.   (*After Whitehouse, Iowa Exp. Sta. Bul.* **192.**)

disease or to anticipation of loss from its increase; (2) by spoilage before the fruit could reach the consumer; and (3) by restricting purchases when scald was most likely to develop and thus affecting market conditions and lessening total consumption.

Records compiled from market reports of car shipments of apples soon after the adoption of oiled wraps showed a reduction of scald in 1922 to 2.8 per cent on the basis of carlots, with a further drop to 0.7 per cent in the next year and an average of 0.3 to 0.4 per cent or even less since that time.

Etiology.—Apple scald is a nonparasitic or physiological disturbance due to the production, by the tissues of ripening or aging fruit, of volatile esters which have a toxic effect upon the tissues if conditions are such that they accumulate in the tissues or in the surrounding air. This conclusion is based on the fact that typical scald can be "artificially produced in a few days' time by exposing apples to the vapors of ethyl acetate, amyl acetate or methyl butyrate" and on the protecting effect of circulating air or of substances which can absorb these volatile products and so prevent their accumulation. It was suggested at one time that the scalding might be due to a shortage of oxygen or to an excess of carbon dioxide, because of the massing of large quantities of fruit during storage, but it has been shown that scald will develop when there is no shortage of oxygen and not be produced by an excess of carbon dioxide.

The time of appearance and the severity of the trouble are influenced by seasonal and orchard conditions and by the environmental influences that operate during packing, transportation or storage or on the market. The susceptibility to scald is influenced by (1) maturity and color of the fruit at picking time; (2) the amount of moisture available during the growing period; and (3) the size of the fruit. Well-matured and highly colored fruits scald less than immature or poorly colored fruits. Fruit that is picked green may show twice as much scald as well-matured, but not overripe, fruit. Apples are increased in susceptibility by abundant moisture either from irrigation or from natural rainfall. The forcing that may follow heavy irrigation has been known to cause three times as much scald after the fruit was removed from storage as in fruit from lightly irrigated trees subjected otherwise to the same conditions. Large apples are generally more susceptible to scald than small ones, mainly because of a forced growth, immaturity and poorer color.

The development of scald is influenced by (1) the temperature to which the fruit is subjected after picking, during storage or during transit to market; (2) the aeration or ventilation of the fruit; and (3) the humidity of the air to which the fruit is exposed.

The harvested apple is still carrying on its physiological processes and will continue to do so as long as its tissues are living. It must be evident that these processes, which are essentially chemical reactions, will be speeded up by high temperatures or slowed down by low temperatures. Since scald is a chemical phenomenon, it is but natural to expect that temperature conditions which retard respiration and tissue activity in

general would delay the appearance of the disease. Storage at low temperature does not prevent scald, but it delays its onset, simply by slowing down the life processes. The influence of temperature upon scald may be illustrated by the results obtained by numerous investigators that immediate storage at 32°F. resulted in more delay in the appearance of the disease than storage at any higher temperatures, especially if the fruit was in prime condition at picking. With fruit that has reached prime maturity at picking time, the appearance of scald is advanced by delayed storage or by holding at temperatures higher than the storage temperature. Immature fruit is reported to scald less by holding at ordinary temperatures until it reaches prime maturity before placing in cold storage.

Stagnation of the air is conducive to the development of scald; hence piling apples in large heaps, close ranking of containers, use of tight containers or storage in poorly ventilated warehouses or storage rooms will favor the onset of the disease. If delayed storage is necessary, it is important to provide conditions which will give the best possible circulation of the air, as by this means the incidence of the disease will be delayed when the fruit goes into cold storage. The importance of aeration in the storage room is emphasized by the fact that scald is often less in well-ventilated cellars and air-cooled storage rooms than in commercial cold storage. The circulation of the air is the important feature rather than the introduction of fresh air. It has been noted that apples near the aisles or doors of a storage room are less affected than those in the center of the stacks. Crowding in a poorly ventilated room will give a large amount of scald, but if, in addition, tight containers are used, scald will be still further increased. It has been shown that apples in ventilated barrels, baskets or hampers scald much less than fruit in tight barrels, the difference being due in part to the more rapid cooling in the ventilated packages but mainly to the free access of air. For this same reason, boxed apples scald less than barreled stock, unless the boxes are stacked too tightly to permit a circulation of the air. The early part of the storage period, that is, the first six or eight weeks, is an especially critical time in the storage life of the apple, and, during this period, good aeration and ventilation are essential. It has been shown that scald is increased by high humidity of the storage room, but the ventilation and the temperature have more influence. The maximum amount of scald will develop in immature fruit from heavily irrigated orchards, packed in unventilated containers and held at high temperatures in humid, poorly ventilated storage rooms.

Susceptibility of Varieties.—Under unfavorable conditions, scald may occur on almost any variety of apple, but certain varieties are especially susceptible. It is generally agreed that green and yellow are

more susceptible than red varieties, although in some red varieties the disease becomes of commercial importance. Partially colored fruits scald mainly on their green or yellow surface. Grimes, Mann, Tolman Sweet, Winter Banana, Yellow Bellflower, Rhode Island Greening and Green Newtown were listed as the most susceptible yellow varieties and Baldwin, Gano, Missouri Pippin, York Imperial and Winesap as the most susceptible red varieties, on the basis of storage studies in New York. Grimes, Mammoth Black Twig, Sheriff, Northwestern Greening, Willow Twig and less commonly Winesap were listed as the principal varieties on which scald is commercially important in Iowa. Other varieties which have been listed as especially susceptible are the Rome Beauty, Granny Smith, Stayman Winesap, Wagener, Baldwin and Wemmershoek (South Africa).

Prevention.—While the scald of apples cannot be absolutely prevented, it can be held in check to such an extent that market losses can be nearly eliminated. While some few practices have proved of outstanding value, the progressive fruit man will give attention to both preharvest and afterharvest factors. The following control features should be kept in mind and followed as closely as possible to secure the production and marketing of a high-grade product: (1) Allow fruit to reach prime maturity before picking, and follow cultural practices to produce the best color possible for the variety. Avoid, especially, undermatured or overstimulated stock except for early consumption. (2) Avoid delayed storage of apples that have reached prime maturity, but if delay is necessary provide for free access of air during the prestorage period, and protect as much as possible from the direct sun or high temperatures. Hold immature fruit until it approximates prime maturity before putting into cold storage. (3) Store fruit as quickly as possible after picking, and reduce the temperature of the *stored fruit* to 32°F. (permissible range 31 to 37°) and the relative humidity of the air in the storage room between 80 and 85 per cent. In some localities certain varieties, such as the Yellow Newtown, for example, may require a slightly higher temperature (36 to 40°) to prevent "internal browning." (4) Arrange for as thorough and complete aeration of the storage room as possible, keeping in mind that the movement of the air is more important than the introduction of fresh air. (5) Avoid the use of tight containers, and provide open stacks in the storage room. The use of ventilated or open containers helps to retard the appearance of the disease. (6) Practice wrapping of the fruit, using special oiled-paper wrappers. Almost complete control is obtained with the use of the oiled wrappers as first reported by Brooks, Cooley and Fisher in 1923. The injurious volatile esters thrown off by the apple are absorbed by the oil of the wrapper in the same manner that butter or other fats take up various odors, and thus are prevented from acting

while heat canker occurs "somewhat uniformly in the northern Great Plains area and causes a marked loss in flax production." This disease has also caused very heavy losses in a number of localities in Ireland (1940, 1941).

Symptoms and Effects.—Heat canker is generally first noticed in plants breaking over at or near the ground level "as though whipped off by the winds or gnawed by insects." This effect is due to the killing of the cortex of the stem above the ground line, while the plants are still young and tender. If this killing occurs when the plants are less than 3 inches high, the young plants generally wither and die. If the plants are 3 to 5 inches high at the time of the injury (rarely, if taller), they may fall down but remain alive for an extended period. More mature plants may show basal cankers, generally with an enlargement of the stem above the lesion. The affected plants may die as a direct result of the girdling of the cortex or the weakened stem may be severed by winds or by the action of saprophytic fungi.

Fig. 233.—Flax seedlings affected with heat canker. (*After Reddy and Brentzel, U. S. Dept. Agr. Bul.* **1120**.)

The marked constriction of the stem within the limits of the canker is due to the death and shrinkage of the cells of the cortex, while the enlargement above the canker is due to the interruption of the downward movement of elaborated food which can no longer be carried to the root system. In case the enlarged portion of the stem comes into contact with moist soil, adventitious roots may start, but under dry conditions they do not form.

Losses have been caused by the heat canker varying from slight to heavy, with almost complete destruction of fields in isolated cases. In the experimental plots at Fargo, N. D., the percentage of cankered plants when unshaded ranged from 37.2 to 46.

Etiology.—The heat canker of flax is the result of the high temperatures of surface layers of dry soil that are in immediate contact with the tender tissues of succulent young stems. The amount of the injury is apparently influenced by the compactness of the soil, the succulence of the tissues and the absolute temperature. In the experimental plats (Fargo) during 1920 and 1921, there were many days when the maximum

soil temperature at a depth of ½ inch varied from 40 to 50°C. (104 to 122°F.), while the temperature at the surface was in many cases 4.5 to 7°C. higher.

The nonparasitic nature of the disease was indicated by the entire absence of any organism in young cankers and by the failure of any isolants from older lesions to reproduce the disease by inoculations. Field observations and tests in experimental plats gave evidence in favor of

FIG. 234.—Basal portion of plants shown in Fig. 233 enlarged to show constricted areas and enlargements. (*After Reddy and Brentzel, U. S. Dept. Agr. Bul.* **1126**.)

heat and light being the important causal agents. Some of these relations were as follows: (1) canker was more severe in rows with wide than with close spacing and also more destructive in thin than in thick sowings; (2) a growth of weeds seemed to lessen the amount of the disease; (3) the shading by a cereal nurse crop had the same effect in reducing the canker as a weed crop; (4) providing partial canvas shade reduced the disease or prevented it entirely; (5) the canker was less when the soil was covered with a yellow sand to a depth of ¼ inch than on the same heavy, dark soil unprotected by the sand layer; (6) a firm crusted surface soil seemed to favor the disease, while a mellow soil without a crust lessened it in

amount; and (7) a cankered condition similar to heat canker was produced artificially.

Heat canker always appeared following the days when the temperature at the surface of the soil was high. The observations indicated "that under conditions favorable for the production of heat canker, the critical temperature is about 54°C." When moisture and temperature conditions produce a soft, succulent growth of the young plants and this is followed by crusting of the surface and a period of critical temperatures, the heat canker is likely to develop, while the same temperatures will have a less injurious effect on plants which have made a firmer growth. Early seedings sometimes escape the trouble because they may pass the susceptible stage before the advent of high temperatures.

Prevention.—Since the trouble is due to the high temperatures during the early period of growth, heat canker should be controlled either by seeding early, so that the seedlings pass the susceptible stage before the advent of the hot weather, or by adopting cultural practices that will partially shade the young plants and thus hold down the temperature of the surface soil. For this latter purpose, higher rates of seeding and drilling north and south rather than east and west have been suggested.

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CROWN ROT OF TREES

The term "crown rot" or "collar rot" is used to designate a bark disease in which the primary lesions are located in the bark of the basal part of the tree trunk or on adjacent portions of the large roots. Splitting and death of bark are followed by decay, and the affected tree may be partially or completely girdled, resulting in serious derangement of functions or in death.

Symptoms and Effects.—The most striking symptoms of crown rot of fruit trees can be noted during the growing season following the dormant season in which the initial injury occurred. Following are the noticeable features: (1) a scant foliage with leaves of small size; (2) pallor or chlorosis of the foliage; and (3) a sickly coloration of the bark of trunk and limbs, especially in smooth-barked species. In the apple, for example, the bark assumes a reddish-yellow cast which makes it possible easily to pick out the affected trees from a distance. A portion of the tree or the entire tree may show the symptoms of the disease.

The symptoms described are practically certain indications of the presence of well-developed lesions at the base of the trunk or on the larger roots, where bark will be found to be dead and discolored, either on one

side of the trunk or completely encircling it. The bark soon becomes sunken, cracks or fissures may appear, and, during the progress of the season, the dead bark disintegrates and weathers away somewhat. Finally, the underlying wood may be more or less exposed. The injured bark may be largely at the ground line and below, at the junction of large roots with the trunk, or it may be at the ground line and extend 12 to 18 inches or more up the trunk. Especially in portions of lesions protected by the soil, the outer portion of the bark may be left intact while the inner portions may disintegrate into a granular mass which becomes powdery when dry. The wood beneath affected bark may become brownish or even charred in appearance. Lesions may extend gradually, advancing downward most rapidly, less rapidly upward and rather slowly laterally. If bark disintegration is checked, callus may be formed, and more or less healing of the wound may follow. In some cases, all of the bark is not killed down to the cambium and, under favorable conditions, regeneration of tissue may occur in sufficient amount to prevent the development of an open canker.

The final result of crown rot will depend on the depth and surface extent of the lesions and probably also on the soil and moisture conditions which prevail during the season following the initial injury, while the abundance of wood-destroying fungi in the environment may play a part. In the severe type in which the bark is killed down to and including the cambium, serious results are likely to follow. Complete girdling will, of course, prove fatal unless bridge grafting is resorted to, and cankers involving three-fourths of the circumference of the trunk, will cause serious interference with the life of the tree. Cankers involving only part of the circumference may be extended the next season and cause complete girdling, or the advance may be checked.

Etiology.—A survey of the literature concerning collar rot shows that troubles having very much the same symptomology have been attributed to a variety of causes. Most of the evidence points to the fact that the disease in the majority of cases is but one phase of winter injury somewhat akin to winter sunscald and crotch cankers. Collar rot or root rot may, however, occur independent of winter injury, sometimes being due to bacterial invasion (see Fire Blight) or various fungous parasites (see Mushroom Root Rot). In Colorado it has been attributed to arsenical poisoning, while alkaline irrigation water has been looked upon as an inducing factor or as contributing to the prevention of healing after the lesions have been formed by other agents. There is no doubt that wood-rotting fungi play a secondary part by invading the wounds, whatever their primary cause may be.

The conclusion that most cases of crown rot of fruit trees are due primarily to low temperatures and excessive or late fall growth is based

largely on field observations rather than on experimental evidence. It has been very definitely shown that the early stages of the crown rot originate during the winter or dormant season. The first visible stages "consist of discolored and often ruptured tissues variously distributed in streaks and patches in the bark," and this condition can be found in the late winter and spring. The progress of the trouble following the initial injuries is rather varied, and the tissue changes are somewhat complex. The initial injuries may be outgrown, or typical cankers of severe type may develop. The nonparasitic character of the disease is supported by the fact that no organisms of any kind are commonly found in young lesions. The base of the tree trunk matures its tissue more slowly than portions higher up and consequently is susceptible to injury from freezing in the late fall or early winter. Crown rot is, therefore, believed to be an early form of winter injury, in contrast to sunscald, which is a late winter or spring form of injury.

Prevention and Treatment.—There is no possibility that crown rot can be entirely prevented, but attention to cultural and irrigation practices will give some relief. Effort should be made to keep trees from growing too rapidly or too late into the fall in order that they may not be subjected to unfavorable temperature when the bark is still immature. It has also been suggested that varieties of trees which are subject to this type of winter injury should be headed low regardless of the inconveniences which may be experienced in cultivating. The swaying of young trees by the wind is believed to play some part in the production of the initial injuries; hence windbreaks might prove of special value in lessening the disease.

In orchards in which crown rot prevails or when conditions have been such as to indicate the probable occurrence of the disease, the trees should be examined during the spring and early summer for indications of bark injury at the base of the tree trunk. In case such injuries are found, the soil should be removed from contact with the injured portions, and all of the dead bark cut out. The cut surfaces should then be treated with an antiseptic and waterproofed, or a combined antiseptic and waterproofing treatment may be substituted. Waterproofing alone, using coal tar or grafting wax, would be sufficient if one could be certain that no organisms were concerned. In cases of complete girdling or when the lesions involve a large part of the circumference, trees may be saved by resorting to bridge grafting or perhaps in other cases preferably by approach grafting. It has been shown by more recent work that uncovering the injured crown not only offers better conditions for healing following injury but increases the resistance of the exposed parts to freezing injury. This gives support to the idea that injury might be avoided by guarding against too deep· setting of trees when planting the orchard. Moderate pruning and removal of fruit are also believed to facilitate recovery.

References (H. 181–182)

WINTER SUNSCALD OF TREES

Bark injury on any portion of a tree may result from the freezing to death of the tissues, including the primary cambium and the external parts, or in superficial cankers the primary cambium and inner bark cells may escape injury. When the bark injury is localized, more or less well-defined dead areas are formed, which with cracking and peeling of the dead bark may become open wounds or cankers. These cankers most frequently occur on the southwest face of the tree trunk, from the ground up, on the sun-exposed surfaces of large branches or at the junction of scaffold branches with the main trunk. Whenever the freezing injury is

A   B   C   D

Fig. 235.—Diagrammatic cross sections of tree trunks showing superficial, moderately deep and deep sunscald cankers.

localized on sun-exposed surfaces, it is classed as "winter sunscald," a term used to distinguish the trouble from summer sunscald due to the killing effect of high summer temperatures. Sunscald of some thin-barked trees from overheating during the intense sunshine and high temperatures of midsummer, coupled with drying out of the tissues, does undoubtedly occur, but winter injury is probably a much more common cause of cankers, on both fruit and forest trees.

Symptoms and Effects.—The early stages of winter sunscald are frequently not observed. Following the period when the injury occurred, the affected bark may appear dull or discolored, and there may be some shrinkage due to drying out of the tissues. Soon the bark will be found to be loose from the wood, and the brown, dead portions can be readily peeled away from the underlying wood, which may also be discolored. In the more severe types of injury, the bark may split and crack quite soon, or this may be somewhat delayed, but the final result in the undisturbed cases will be the weathering and peeling of the bark to produce more or less of an open wound. In the more moderate types of winter sunscald the injury may be confined to a narrow strip on the southwest face of the trunk, while in more severe cases the bark may be killed for nearly the entire circumference of the tree. The immediate injury will depend on

the extent to which girdling has been completed and the functional activity of the remaining portions lowered. The most severely sunscalded trees may die later in the season; while, in those less severely injured, healing will take place, and the trees may survive if the entrance of wood-rotting fungi can be prevented. This secondary injury from the entrance of wood-rotting fungi is a very fruitful cause of the final decline of trees. The disintegration of the wood by fungi which enter through sun-

FIG. 236.—Old winter sunscald canker on apple. [*After Mix, N. Y. (Cornell) Agr. Exp. Sta. Bul.* **282**.]

scald cankers or crotch cankers may so weaken a tree that it goes down during the stress of windstorms. Lesions may occur on the face of large limbs and also at crotches. Low crotches are more susceptible to winter injury than higher crotches, and narrow ones, more than wide.

Etiology.—It was at one time believed that winter sunscald was due to the rapid thawing of the frozen tissue on the sun-exposed face of the trunk or large branches. The present opinion is that the injury is the result of the direct freezing to death of the tissue. This conceivably might result from one or the other of two ways or by a combination of these:

1. An increased tenderness in the tissue on the sunny side of the trunk in late winter causes it to kill at a temperature not low enough to injure the still dormant tissue of the shaded side.

2. The tissue on the sunny side kills at a higher temperature, due to the more rapid temperature fall that may take place after a cold, sunny day in later winter (Mix, 1916).

As a result of freezing tests carried out by Mix, the conclusion was reached that apple bark on the southwest and on the northwest sides of the trunk showed no appreciable difference in hardiness and that, therefore, the view that the warmth of the sun, by promoting metabolic activities, caused increased tenderness must be eliminated as the cause of sunscald. It is of interest to note, however, that trunk tissue from all sides gradually becomes more tender as the end of the dormant season is approached. This would suggest that sunscald is more likely to occur in late winter or spring than in the early part of the dormant period.

The view that freezing to death is due to a "rapid temperature fall consequent to a warming up of the tissue above freezing by the rays of

the sun on a bright, cold day in late winter" is supported by observations and experiments: (1) the higher day temperature of the southwest side of tree trunks as contrasted with the northeast side varying from a slight excess to a maximum of 20° or more; (2) the equalization of temperatures on both sides during the night periods; (3) the increased killing of buds or twigs known to result from rapid freezing as contrasted with slow freezing. On the basis of these facts, winter sunscald is supposed to be a late-winter injury. It is not the result of increased sensitiveness due to late growth or failure of tissues to mature but is liable to occur any season whenever the rate of temperature fall on the sun-exposed side is sufficiently rapid following the warming up by the winter sun, and the minimum reached is sufficiently low.

Bartlett and D'Anjou pears are very susceptible to winter sunscald. Among apples Newtown, Spitz, Ortley, Jonathan and Winter Banana are reported as suffering more than many other varieties. Most injury is reported on poor soils lacking proper drainage, while it is further increased by excessive irrigation, low fertility and overcropping. As might be expected, the damage is least on north and northeast slopes. Pears on French rootstocks have been reported to suffer less than those on Japanese rootstocks.

Prevention.—In many regions, winter sunscald may be of such rare occurrence that preventive measures are not justified. In those regions where sunscald is frequent, practices may be followed which will prevent the excessive heating of the sun-exposed bark. This may be accomplished in either of two ways: (1) by shading to protect the bark from the direct action of the sun or (2) by modifying the absorption of heat with full exposure. The use of board or lath screens has been recommended with trees that are headed high, while very low heading of trees has been practiced as a protective measure with some success. Board trough screens, 3 feet high and set 6 inches from the trunk, have been reported to reduce winter sunscald in pears from 85.6 per cent to 3.3 per cent. In some tests, painting the trunks with white water paint has given better results than the board screens. It is claimed that some protection may be obtained by planting young trees so that the lowest limb is on the southwest side.

Spraying or painting the trunk and large branches with whitewash has been quite generally recommended for northern regions where winter sunscald is common. The protective effect is due to the fact that the whitewashed bark does not heat up to such high temperatures as normal bark during the warm winter days. In the treatment of sunscald cankers by the removal of all dead and decayed bark, the recognized methods of disinfection and protection of the wounds should be practiced, with care to leave a V-shaped cut at the bottom.

LODGING OF CEREALS AND OTHER CROPS

The lodging or falling down of cereals previous to harvest is a common phenomenon in many regions, while in others it is rather rare.   No single cause of the lodging can be sighted, but the following may be noted as contributing factors: (1) excess of nitrogen, or, at least, a large amount of available nitrogenous food materials, which stimulate the plants to make a luxuriant growth with heavy foliage; (2) an abundance of moisture in soil and air, which promotes a succulent type of growth; (3) frost injury owing to localized killing of the meristematic tissue at the base of certain internodes; (4) the attacks of insect pests or fungous invasions (straw breakers or foot rots), which are clearly parasitic in character or favored by frost injuries or traumatisms; (5) mechanical breaking or lodging due to the direct action of wind, hail or rainstorms.   While one to several of these factors may be operative in any single case, the most common cause of general lodging is a weak development of the bases of the culms or stalks as a result of the lack of light.

Symptoms and Effects.—The lodging due to partial etiolation of the basal portions of the culms involves a weakening and abnormal elongation of the second internode from the base, the lowest stem member being generally too short to bend.   Under conditions which promote the weakened growth, the developing culms may fall, owing to the bending at the second internode.   Lodged grain may partially right itself, owing to renewed growth in other internodes as a result of the geotropic response induced by the prostrate position.   The lodging of the grain interferes with the normal physiologic processes, especially photosynthesis and transport of crude food materials from the root system to aerial parts, and, consequently, the crop is lowered in both quantity and quality. In addition, the lodged grain is much more likely to suffer injury from semiparasitic fungi and from such troubles as powdery mildew and rusts, while the difficulty of harvesting the grain that does develop is the cause of additional losses.

Etiology.—The lodging of cereals was at one time attributed to a lack of silicic acid (H_2SiO_3) in the soil, thus lessening the amount of silica in the cell walls and thereby weakening the supporting power of the culms. Such an explanation seemed possible until it was shown that there is but little difference between the silicic acid content of normal stiff straw and lodged straw and that a very small amount of silicic acid is sufficient for the production of normal plants.   It has also been shown that the lowest internodes of normal plants are poorer in silicic acid than other parts of the plants, which would be opposed to the supporting function of the silicified cell walls.

It has been shown that lodging is due to a modification of the structure of the lower internodes as a result of the interaction of many factors, with shading playing a prominent part.   In thick stands of grain, the numerous culms with clustered and overlapping leaves prevent the penetration of light and also tend to prevent the circulation of the air and thus hold a surface stratum of more humid air.   Both lack of light and

FIG. 237. —Cross sections of normal and lodged rye culms.   (*After Koch.*)

increased humidity of the air promote the formation of delicate or thin-walled tissue of elongated cells, as opposed to the firm growth with well-developed mechanical tissue which is characteristic of well-illuminated structures surrounded by an air of more moderate humidity.   Further support of the conclusion that shading is the most important factor in causing lodging has been obtained by the artificial production of lodging by shading the basal parts of the plants, while the more distal parts were subject to normal light intensity.

The stems of normal cereals show a well-developed zone of thick-walled, prosenchymatous tissue below the epidermis, but in the partially etiolated basal internodes of shaded culms, this cylinder of supporting tissue is poorly developed and other tissues do not have normal supporting power.   The greater length of these shaded internodes and the

thinness of the cell walls make such stems poorly suited to withstand the strains they mùst bear, and they easily go down during wind- or rainstorms.

According to recent studies lodging results: (1) when there is a relatively low content of dry matter per unit length of culm; and (2) such culms are relatively small in diameter and are accompanied by a low carbohydrate-nitrogen ratio, resulting from hypernutrition, shading or high temperatures, the complex giving a relatively high proportion of vegetative growth. Soils rich in available nitrogen and well supplied with moisture predispose to lodging, since tillering is increased and the heavy growth of foliage increases the shading. Thick seeding on moderately rich soil may sometimes lead to very general layering, because of the shading due to the extremely dense stand. With the modern practice of drill seeding in which the rate of seeding can be accurately controlled and adapted to the soil type, the danger of lodging is lessened.

Prevention.—The most important practices which may be followed for cutting lodging down to a minimum are as follows: (1) careful attention to the rate of seeding to suit the soil conditions so as to produce a stand which will allow penetration of light and a free circulation of the air; (2) attention to cultural practices, including rotations and the use of fertilizers so that excess of available nitrogen will not be offered; (3) the selection of varieties suited to the environment. Stiffness of straw is affected by length and diameter of internodes, thickness of the culm wall, the number of vascular bundles, the width of the lignified tissue in cross section and the thickness of the sclerenchyma cell walls. The ability to stand up will vary, therefore, with the variety, and improvement in this respect is one of the problems of the cereal breeder.

Other crops besides cereals frequently suffer from lodging, for example, field peas. Their stems are naturally rather weak; and when they do go down, rotting may cause additional damage if there is an abundance of moisture. In localities where the lodging tendency is very pronounced, a supporting crop is sometimes mixed with the peas. It is not uncommon to have too heavy stands in seedbeds in the open or under glass. Under such conditions, the hypocotyl frequently elongates to several times normal length and the young seedlings fall over, and, as a result, irregular, twisted stems are formed. The partially etiolated stems are then more readily infected with damping-off fungi.

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CHAPTER XX

DISEASES DUE TO MANUFACTURING OR INDUSTRIAL PROCESSES

As a result of modern conditions in cities and the proximity of various types of industrial concerns, cultivated plants and native vegetation are frequently exposed to unfavorable factors which may operate either through the air or the soil environment.

Electrical Injuries.—The development of electric-light systems, trolley lines and high-power transmission lines has introduced a new element of danger to the trees of towns and cities. It is a matter of common observation in many sections that lightning causes much injury to trees, while numerous cases of lightning injury to field crops are on record. It is perhaps not so generally understood, however, that trees may suffer from electric discharges from transmission lines when these are too close. Either alternating or direct currents may cause injury, but the former is apparently less injurious. An electric current acting on a plant at certain strength—the *minimum*—may cause just perceptible stimulation, while the *optimum* causes the greatest stimulation. Beyond the optimum, plant activities are retarded, and at *maximum* strength death ensues. The maximum current necessary to cause death is exceedingly variable.

As a general principle, trolley or electric-light wires should not be allowed to come in direct contact with the trunk or limbs of trees. If, however, contacts cannot be avoided, proper insulation of the wires should be provided.

Injury from Dusts.—Materials in dust form may be produced by manufacturing or industrial processes and fall on vegetation or surrounding soil and cause injury by sooty deposits on leaves, effecting a reduction of photosynthetic activity, or on berries, which may thus be ruined for market; and by mechanical interference with the physiological activities, or by their ultimate toxic action. Toxic materials may be dissolved in the soil water, or they may become insoluble by interaction with soil constituents. Metallic dusts that are soluble in water or in water containing a small amount of carbon dioxide may be toxic to foliage or other plant organs when wet with dew or rain. Injuries have been recorded from many different chemical elements or their compounds or from mixed dusts, including zinc, copper, aluminum, lead, arsenic, salt, soda and oxalic acid, which have been shown to act either directly on aerial parts or through the soil; also from the dust from cement mills, limekilns, magnesite roasters, the processing of coal and the manufacture of briquettes,

and from soot produced by imperfect combustion and even from tarred or asphalt-treated roads.

Injuries from Toxic Gases.—Various gaseous materials may be set free in manufacturing or industrial processes, and these may diffuse through the air or the surrounding soil and reach the aerial parts of plants or their root systems either in gaseous form or dissolved in dew, mist or raindrops. Mention may be made of sulphur dioxide, fluorine compounds, hydrochloric acid, chlorine, sulphuric acid, nitric acid, ammonia, tar and asphalt gases, illuminating gas and also of numerous materials of minor importance. These may be by-products of industrial plants, as sulphur dioxide from smoke and smelters, or the main output of the plant, as illuminating gas. Very high toxicity to plant life, even when present in only minute quantities may be noted especially for sulphur dioxide and illuminating gas or its principal constituent, ethylene.

Smoke Injury.—The atmosphere of industrial centers in the large cities and surrounding smelters in outlying districts is frequently polluted with various gases or dust materials which have injurious effects upon neighboring vegetation. The most important sources of injury are the products of the incomplete combustion of coal and the gaseous or solid wastes resulting from the smelting of ores, but the fumes from railroad centers and central heating plants are of local importance, especially to the more sensitive evergreens. Investigations have shown that dust and metallic fume are elements of minor importance as far as damage to vegetation is concerned, in comparison with the gaseous constituents of smoke. Under open or field conditions, the most injurious gas is sulphur dioxide (SO_2), which is formed in the burning of coal and in the smelting of sulphide-containing ores. Smoke injury as here considered will then be confined very largely to the effects of sulphur dioxide.

In this country, the smoke nuisance in a number of the larger cities has prompted special investigations, while the smoke problems of English industrial centers like Leeds and Sheffield have been given careful study. The injury from smelter fumes has been a problem in various portions of the United States and has attracted the attention of both chemists and plant pathologists.

1. *Symptoms and Effects.*—Three different types of injury from sulphurous acid in the air are recognized: (*a*) *acute,* when the amount of gas is abnormally high, being characterized by the rapid bleaching or disappearance of chlorophyll and in most severe form by the death of the entire plant; (*b*) *chronic,* when small quantities of sulphur dioxide are generally present, leading to a general depression of physiological processes, including photosynthesis, metabolism, cell division, etc., with retarded growth, exhausted food reserves, failure to blossom and set fruit, early leaf fall in deciduous forms or fall shedding of leaves by ever-

greens, ending ultimately in death; and (c) *invisible*, or the reduction of growth increments not visible to the naked eye but expressed by yields or by modified composition shown by careful measurements or by chemical analyses.

Fig. 238.— Beech leaves showing SO_2 injury. (*After Schroeder and Reuss.*)

There is no hard and fast line between acute and chronic injuries, but acute injuries are first indicated by characteristic changes of the chlorophyll-bearing structures. The response is somewhat different for the conifers, deciduous trees and shrubs and herbaceous forms. Acute injury in many *conifers* is marked by a wine-red coloration of the needles, sometimes for their entire length or sometimes first at either base, tip or middle. The needles then turn brown, shrivel and fall if the action of the gas continues or is sufficiently severe. The amount of discoloration and death of leaves is variable, but, in general, their length of life is shortened,

and trees close to sources of smoke frequently retain only the needles of a single year.   In *deciduous trees and shrubs*, the most common result is the appearance of yellowish-brown to dark-brown dead areas in the inter-costal areas of the leaves, while the mesophyll adjacent to the prominent nerves remains green for the longest time.   Because of this localization of the dead areas, the leaves showing various types of venation will exhibit

Fig. 239.—Maple leaf showing SO₂ injury.   (*After Reuss.*)

quite bizarre color patterns.   While the intercostal location of the dead areas is the most frequent, the injured leaves may sometimes either show a dead brown border or the discoloration may be confined first to either tip or base of the leaf blade.   In species of Prunus or other forms showing a natural tendency to shot hole, the dead areas may fall away, leaving perforations.   In *herbaceous plants*, the injured tissues may show all gradations of color from reddish brown or almost black to light yellow or straw color, with the lighter colors predominating.   Injury to wheat before blossoming time is marked by the reddening of the leaf tips, which later turn yellow and finally become almost white.   Other cereals and many grasses may show only a yellowing and bleaching of the leaves, beginning at the tips.   Alfalfa and related legumes may show extremes of bleaching or a clean, white appearance of the affected parts, the dis-

coloration advancing from the tips, or margins, of the leaflets so that an irregular green area may be left along the midrib. Lupines may show a dark-brown or almost black discoloration beginning at the tips of the leaflets, the sugar beet may show reddish-brown discoloration with a tendency to appear in the intercostal areas, while the potato may show reddish-purple tints similar to those characteristic of leaf roll. It should be pointed out in this connection that the various leaf discolorations which accompany sulphur dioxide injury are in no way diagnostic characters, for very similar effects may follow injury from other agencies, such as drought, frost, sunscald, etc. Flower parts that have been killed or blighted may resemble those injured by frost, and nectar fluid may be soured, and for this reason be avoided by pollinating insects.

Chronic injury was first recognized for conifers, but the symptoms are not clearly defined, discolorations of leaves being a minor symptom. The recorded effects are: (1) a shortening of the life of the needle leaves; (2) low increments of growth as marked by narrow annual rings; and (3) staghead, or bare terminal branches. In smoke zones, the life of spruce needles may be two to three instead of four to five years, and the life of fir needles may be reduced to four to five years in chronic smoke injury as compared with ten to twelve years in normal trees. The exact effects will, of course, vary with the concentration of sulphur dioxide to which the trees are exposed and with other modifying factors.

Crops like wheat, barley and sugar beets grown in a smoke zone show some foliage injury, reduction in size and vigor of plants, premature ripening, reduced yields and, in barley and potatoes, a lowered starch content and, in sugar beet, a reduced storage of sugar. Beets and other root crops subject to sulphur dioxide (SO_2) have a reduced value for the production of a seed crop the next season. Chronic injury is well illustrated in the industrial areas of Leeds, England: bulbs in some portions flowered the first year, but would not bloom the second season or thereafter; lettuce and cabbage would grow but would not head; the color of flowers became paler or streaked with other colors; and broad-leaved trees and shrubs were dwarfed and suffered early defoliation. The behavior of the common privet in the Leeds area is of interest: 3 miles north of Leeds, it is evergreen and flowers; 2 miles north, it is still evergreen but does not flower; 1 mile north, but a few of the leaves are retained during the winter; while in the center of the city the leaves fall in January and in the heart of the industrial center in November. Chronic injury has been reported for tree fruits and grapes in other areas. Reduction in set of fruit may result from the action of sulphur dioxide on the maturing of anthers and pollen or from pathological changes in stigmas, styles, egg apparatus or even young embryos.

The existence of *invisible injury* has been disputed by some investigators, but it has been pointed out that serious injuries may occur in the absence of either acute or chronic symptoms. The action of the sulphur

dioxide has a depressing effect on photosynthesis and other physiological activities, resulting in a general slowing down of constructive metabolism. Some of these depressions are reflected in the lowered starch content and reduced weight of cereals, reduced sugar content of organs in which this carbohydrate is normally stored, a low rate of protein to nonprotein sulphur, reduced or lowered viability of seed and decreased hardiness or increased susceptibility to the inroads of some parasites. Oats grown in the outlying districts of Leeds had a germination of 98 per cent, while seed from the industrial center showed only 17 per cent viable. The effect on hardiness of winter annuals is shown by the winterkilling of spinach, cabbage and wallflower in the regions around Leeds in which the annual deposit in soot per square mile was 200 tons or more, whereas beyond this area in regions with an annual deposit of 100 tons per square mile fall planting was uncertain, while the same plants were winter hardy in the more outlying districts.

The indirect effect of smoke pollution as a result of soil changes has been emphasized by some investigators. That the acidity of the smoke will affect the number and activity of the soil bacteria, especially the nitrifying forms, has been shown by bacterological analyses. Long-continued exposures of fields to smoke fumes have been shown to cause injury by the increase of soil acidity, and deliming effects; also by a reduced formation of fibrous roots and root hairs.

2. *Etiology.*—It has been repeatedly demonstrated that sulphur dioxide is the most important polluting agent in the smoke of industrial centers or from smelters, and experiments have shown the extreme toxicity of this compound for growing plants. Sulphur dioxide is a colorless gas, with a characteristic suffocating odor, and is 2.21 times heavier than air. It has a bleaching action upon many organic coloring matters, as may be illustrated by its effect upon chlorophyll and the pigments of flowers. In the presence of water, it behaves as though *sulphurous acid* (H_2SO_3) were formed, but this substance has never been isolated. It may be still further oxidized to form sulphuric acid (H_2SO_4), and some of the spotting of leaves and flowers in smoke zones is due to the action of this acid.

The incidence and amount of injury depends upon (*a*) the concentration of sulphur dioxide to which the plants are exposed; (*b*) the duration of the action; (*c*) the time of action with reference to the period of illumination; (*d*) the humidity and temperature of the air; and (*e*) the degree of turgidity of the leaf tissues.

The average person cannot detect sulphur dioxide in the atmosphere by the odor when the amount is below 3 parts per million of air. Injury to plants may result, however, when the amount is much less than can be detected by odor. The concentrations required to do injury to plants,

according to published reports based on field analyses and experimental tests, vary from 1 to 40 parts sulphur dioxide per million parts of air. The toxicity danger point will vary for different plants and will be affected by their stage of development but will also be modified by environmental factors. The toxic limits for some trees have been given as follows: oak, 1 to 720,000; pine, 1 to 500,000; and beech, 1 to 314,000. Roses show visible injury with concentrations of 1 to 250,000 to 500,000. It is claimed that a concentration of 10 parts sulphur dioxide per million parts of air is necessary to produce injury to growing grain. The figures are sufficient to emphasize the extreme toxicity of sulphur dioxide.

The injurious effect of sulphur dioxide on mature foliage is due to its diffusion through the stomatal openings into the interior intercellular spaces and its penetration into the living cells, where it interferes with the physiological functions of cytoplasm, kinoplasm and chlorophyll or, in acute injury, completely inhibits the life processes. That the entrance is through the stomata is shown by the fact that any treatments which cause a closing of the stomata will either prevent sulphur dioxide injury entirely or very greatly reduce it. This injury depends, in part, on the affinity of the sulphur dioxide for oxygen and its tendency to combine with aldehydes, substances formed especially in chlorophyll-bearing cells. Sulphur dioxide will be expected, therefore, to have a profound influence upon the photosynthetic processes, or the construction of carbohydrate food. Microscopic tests show that leaves injured by sulphur dioxide will contain either no starch grains or very few, and this is in accord with the influence of sulphur dioxide in reducing the carbohydrates in seeds or vegetative storage organs. The reduced supply of available carbohydrates will, therefore, materially reduce the constructive processes or the growth of the injured plants.

The presence of small quantities of sulphur dioxide has a marked effect on the process of transpiration, or water loss. First, there is an increase in the rate of transpiration, to be followed soon by a lower water loss than takes place in normal plants. Since transpiration is something of a measure of growth, retarded growth should be expected in sulphur dioxide poisoning.

Sulphur dioxide injury is increased by light, moist air and high temperature. When plants are exposed to a toxic concentration of the gas, specimens placed in the light are injured, while others treated in every way the same, except for being kept in darkness, will show no injury. The amount of injury for a given concentration of gas is proportional to the intensity of the illumination, or it might be stated perhaps with more exactness that the greater the photosynthetic activity the greater the injury. This being true, the sulphur dioxide injury is negligible during the night periods and sinks to a minimum during the winter when plants

are dormant. It has been demonstrated frequently that injury occurs sooner in warm, moist weather than under cool, dry conditions.

3. *The Diagnosis of Sulphur Dioxide Injury.*—The determination of the presence or absence of sulphur dioxide injury in a suspected case is not a simple matter, since symptoms alone are not conclusive proof of the type of injury. The absence of known parasitic troubles should be given consideration, but their presence should not be taken too seriously as indicating the absence of sulphur dioxide injury. In connection with the symptomology, certain tests may be employed which will aid in arriving at a correct diagnosis. Some of these are: (a) an analysis of the air at representative stations for its sulphur dioxide content for comparison

Fig. 240.—Section showing effect of SO₂ on sublenticular tissue. (*After Dr. Kupka.*)

with known toxic concentrations; (b) analyses of the leaves of plants from smoke zones for their sulphite or sulphate content, for comparison with similar leaves from normal habitats; (c) the behavior of special plant indicators, or the use of catch plants. A number of other tests, such as staining with methylene blue, the modification of lenticel structure, and the phaeophytin test of injured leaves, at first thought to be diagnostic have since been shown to hold for other types of injury.

Numerous analyses of smoke-injured foliage have shown that the sulphur dioxide content is frequently two to four times greater in the injured leaves than in normal leaves of the same species. Since, however, the sulphur dioxide content of plant tissues is influenced by the soil composition, conclusions based on leaf analysis must be made with care. Several different cultivated plants have been recommended as catch plants for cultivation in areas in which smoke injury is suspected: (a) beans (*Phaseolus vulgaris*) by Sorauer; (b) species of Polygonum or Rheum by Haselhoff and Lindau; (c) a variety of grapes the leaves of which turn

red when injured by sulphur dioxide (Weiler); and (*d*) *Lupinus angusti-folius* by Stoklasa. The chlorophyll of lupine, which is especially sensi-tive, is decomposed by 0.0004 to 0.0008 volume per cent of sulphur dioxide. The essential in the catch-plant method is the use of a sensitive species in which injury may be determined with certainty by botanical or chemical means.

4. *Susceptibility of Species to Sulphur Dioxide Injury.*—Plants may be grouped as very susceptible, moderately susceptible or resistant to sul-phur dioxide injury. The following are selected from many herbaceous plants tested as typical of the groups: (*a*) *very susceptible:* lupine, clover, beans, peas, lentils, alfalfa and roses; (*b*) *moderately susceptible:* grasses and cereals; (*c*) *resistant:* beets, potatoes, Brassica species, iris and chicory. Lupine is listed as the most susceptible and chicory as the most resistant. Among trees most conifers are very susceptible, most decid-uous trees moderately susceptible, but maple and yew are resistant. Spruce and yew represent the extremes. One authority lists the ash (*Fraxinus excelsior*) as the most sensitive of 30 species of trees.

5. *Control or Prevention.*—The prevention of injury to the natural vegetation or to cultivated crops is largely in the hands of the agencies which are responsible for the smoke production. The methods for the mitigation of the smoke nuisance are based on either the retention of the injurious substances or their dissipation in such dilute form that the con-centration will never reach the danger point. Some of the suggested methods of retaining the noxious gas are condensation methods, decom-position with hydrogen sulphide with the deposition of sulphur, absorp-tion of the acid gases by basic materials or washing the gaseous output with water. If the commercial demand justifies, the sulphur dioxide output may be utilized as a source of sulphuric acid. The devices for diluting the sulphur dioxide before it reaches vegetation are high smoke-stacks, numerous small stacks or some modification of this principle or special devices for diluting the sulphur dioxide with air or combination of diluting and deacidifying. When it can be proved that industrial plants are responsible for crop damage as a result of sulphur dioxide or other exhalations, they are liable for damage.

When the elimination of the smoke injury is not or cannot be accom-plished, a certain measure of relief can be obtained by the selection of more resistant species for cultivation.

Injury from Illuminating Gas in the Soil.—Herbaceous or woody plants growing in streets, yards or greenhouses may be injured by the leakage of illuminating gas into the soil. The greatest amount of damage occurs in shade trees of street or lawn. Injury to trees from leakage of gas into the soil from defective joints or broken pipes is a problem with which every large gas-producing company must contend. Much of the

gas that is manufactured is unaccounted for, part of the discrepancies being due to defective meters or incorrect readings, but more or less actual leakage does occur. Slight leakage of gas into the soil through a long period may cause a slow poisoning of the roots of near-by plants, while, with more pronounced leakage, acute injury may follow very rapidly.

The effects of illuminating gas are variable, depending on the age and stage of development of the plant, the variety or species concerned and the duration of action and concentration of the gas. Some of the effects of illuminating gas in the soil are on *leaves:* discoloration, stunting,

Fig. 241.—Large elms killed by escaping illuminating gas, 1½ years after leakage occurred. (*After Stone, Mass. Bul.* **170.**)

curling, drying, wilting, abscission, and epinasty, or downward curvatures; on *roots:* discoloration, swelling, stunting, proliferation, bending and necrosis or death; on *stems, trunks or branches:* proliferation, cracking, sap exudation and partial or complete necrosis; on *seeds:* inhibition of germination. The production of proliferation tissue by roots is a feature which may be used in diagnosing gas injury, since root hypertrophies occur when the foliage shows no effects. The cortex is sometimes three to four times the thickness of normal roots. This feature might be lacking if the tree was killed suddenly by a high concentration but might appear on roots of adjacent uninjured trees or shrubs. It may be noted that these responses to illuminating gas may be in the nature of *stimulation,* as illustrated by an acceleration of the early development of latent buds, the epinasty of leaves, or the hypertrophy of root parenchyma, while the other effects range from various degrees of *injury* of tissues or organs to killing. It should be noted that most of the symptoms and effects of gas injury are of a general character such as would result from the failure of the root system to function from other causes such as asphyxiation, freezing injury, collar rot, etc.

The injury to roots from the leakage of illuminating gas into the soil has been attributed to: (1) the displacement of the soil oxygen, resulting in an asphyxiation of the tissues; and (2) the direct toxic action of gas constituents. It is doubtful if the asphyxiation of tissues is any more than of minor consequence under open field conditions. Illuminating gases are of variable composition, but they always contain substances which are toxic to roots. The components which have received most consideration as possible toxic agents are carbon monoxide, constituting 20 to 40 per cent of illuminating gas, ethylene, one of the unsaturated hydrocarbon gases, and hydrocyanic acid. Controlled tests have shown that carbon monoxide will produce many of the symptoms of gas poisoning, but the concentration required is 5000 times greater than required for ethylene. Ethylene alone will produce practically the same effects as a composite illuminating gas. Tests have shown that the minimum quantity of cyanide gas causing collapse of tomato stem was the same as the amount present in illuminating gas causing the same response. According to this concept the cyanide is the main cause of the injury from illuminating gas when operating through the root system. This theory was first advanced by Wehmer in Germany and has been supported by American workers.

The certain diagnosis of gas injury is rather difficult and requires the services of an expert. The detection of one of the important constituents of gas, ethylene, in the soil, combined with the characteristic injuries, is of rather diagnostic value. The etiolated-sweet-pea-seedling test will reveal the presence of gas in a soil when no odor is distinguishable. Seedlings may be grown in Petri dishes until several centimeters high and then placed under inverted cans on the soil supposed to contain illuminating gas. Strong concentrations of gas will cause a cessation of growth, while minute traces of it will induce diageotropic growth of the epicotyls; that is, they will grow in a prostrate or horizontal position. It is claimed that this test exceeds many fold the delicacy of any chemical test.

The prevention of leakage of gas into the soil is given special attention by gas manufacturers, since corporations are called upon frequently to pay damages for killed trees. When gas leakage is discovered, it should be stopped at once. Trees, except conifers, already visibly affected are not likely to recover, but any possible recovery will be supported by digging up the soil so as to allow the escape of the gas. New trees should not be set in the place of gas-injured ones until the soil has been thoroughly aerated.

Injury from Illuminating Gas in the Air.—For plants grown in the open, illuminating gas is not likely to accumulate in the air in sufficient amount to cause injury by direct action on the aerial parts, but severe

injury has been noted in house and greenhouse culture. It has been shown that ethylene gas is the poisonous constituent, causing injury when present in very minute quantities. Carnations are especially sensitive, buds being prevented from opening by 1 part of ethylene to 1,000,000 parts of air, while 1 part of ethylene to 2,000,000 parts of air may cause open buds to close. Under house or greenhouse conditions, only small quantities of gas are likely to be present in the air, but many glasshouse plants may be injured by these small quantities of gas. According to recent studies (Krone, 1937) natural gas is very much less toxic than artificial gas.

The types of responses induced by illuminating gas or by ethylene alone are as follows: (1) retarded growth; (2) yellowing and falling of leaves in dicots and abnormal curvatures in monocots; (3) rigor and loss of irritability; (4) flower buds may fail to open or open and drop their petals (roses) or open flowers may close and blight; (5) the formation of soft, spongy proliferation tissue at lenticels or leaf scars or on extended areas of stems; (6) the forcing of latent buds (roses); and (7) the *epinasty* of petioles or leaves, causing drooping, bending, rolling, coiling, the response varying with the species or variety, age of leaves or rate of growth. In some cases complete spiral coils may be formed. In addition to the pathological effects, a short treatment with 20 per cent illuminating gas has been shown to have a stimulating effect upon dormant woody plants, causing them to open buds and produce leaves.

The presence of illuminating gas in a greenhouse may be demonstrated by the use of test plants, such as tomato, scarlet sage, sensitive plants, castor bean or Jimson weed. Healthy test plants of the selected variety may be placed throughout the greenhouse and left for 24 to 48 hours with poor ventilation. Fifty parts of illuminating gas per million of air will cause epinastic growth of the petioles; with greater concentration, the bending down of the leaves will increase; and they will be dropped with a concentration below the limit of the odor of gas. The etiolated-sweet-pea-seedling test previously mentioned may also be used.

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CHAPTER XXI

DISEASES DUE TO CONTROL PRACTICES

The treatment to save crops from losses due to diseases or to insect pests is sometimes as productive of injury as the disease or pest. In treating seeds or plants with either fungicides or insecticides, by spraying, dusting, steeping or fumigating, chemical elements or compounds are employed that are poisonous or toxic to fungi, bacteria or insects, and these same preparations may be toxic or have injurious effects upon our crop plants or upon the commercial products.

Types of Injuries.—Most of the injuries to be charged to control practices result from chemical agents used for sprays or seed disinfectants or from the action of heat or of refrigeration. The following are the principal types of injury: (1) *Leaf injuries:* reduction in size, staining, spotting, edge burn, scalding, general necrosis, shot holing, rolling or curling, pallor of leaves, "yellow leaf" or nearly a complete chlorosis and leaf fall or defoliation. (2) *Blossom injuries:* blighting of parts, failure to set fruit, and failure to set fruit buds for next year. (3) *Twig injuries:* general discoloration, spotting, cankers, gummosis and dieback. (4) *Fruit injuries:* poor coloration, reduction in size, staining, localized or diffuse russeting, spotting, blossom-end or calyx burn, malformation and cracking, dropping before mature, internal necrosis, modified chemical composition and excess of lead or arsenic even after cleaning. (5) *Entire-plant injuries:* dwarfing, general depressing effect, shortened growth period, general necrosis and death of annuals or killing of perennials, increased susceptibility to frost injury. (6) *Root-crop injuries:* reduced yields from soil sterilization (potato from sulphur), reduced yields (potato from lime-sulphur spray), reduced germination of tubers, retarded growth. (7) *Seed and seedling injury:* complete failure to germinate, reduced percentage of germination, malformed seedlings, retarded growth of seedlings. (8) *Acceleration or retardation of physiological processes:* lessened photosynthetic activity, increased respiration, increased transpiration, and lessened annual increments of wood. (9) *Injury to processed fruit:* the production of "springers" or swelled cans with fruit or vegetables sprayed with lime-sulphur.

It should be noted that many of these injuries are similar in general characters to the symptoms and effects of parasites.

Bordeaux Injury.—Through the work of the U. S. Department of Agriculture, Bordeaux mixture was introduced into this country in 1887.

It soon came to be the generally accepted fungicide for the protection of growing crops from the attacks of various parasitic fungi.   The principal complaints of injury from its use have been from orchardists.   Apple and peach trees have been most generally affected.   The injury from Bordeaux has been known under such names as "Bordeaux scald," "spray injury," "Bordeaux burning," "spray russeting," "cork russeting" and "yellow leaf."

1. *Symptoms and Effects.*—The principal injury from Bordeaux is to the fruit and the foliage.   The injury to the *fruit* appears first as small dark or brown specks, less than 1 millimeter in diameter, and these are more or less isolated, or they may be so numerous as to coalesce and form

Fig. 242.—Bordeaux injury.   *A*, severe Bordeaux injury of half-grown Baldwin apples; *B*, Bordeaux injury on a Rhode Island Greening apple.   (*After Hedrick, N. Y. (Geneva) Exp. Sta. Bul.* **287.**)

rather extended russeted areas.   The location of the injured areas will depend upon the position of the fruit at the time when the spray was applied, being on the surface to which the greatest quantity of the spray material adheres.   Severe injuries to young fruits may cause more or less distortion in shape, owing to localized atrophy or shrinking of tissue or in other cases to teatlike malformations.   As the severely affected fruits grow older, deep cracks may form, and these may be healed over with the formation of cork cells.   The appearance of minute red spots, centering at the lenticels, on yellow-skinned apples has been attributed to the effects of Bordeaux, although other agencies may also produce similar effects.   Moderate russeting is of main concern as affecting the appearance of the fruit, but the market value may be ruined by the more severe types of injury.   Bordeaux-injured apples have poor keeping qualities, since they lose moisture more rapidly than normal fruits and are more easily invaded by rot-producing fungi.   A very similar russeting of the

fruit is caused by frosts during the young stages of growth (see Frost Injury, page 514). On the foliage, Bordeaux may cause a contraction of cell walls and an agglomeration of cell contents; sometimes a sudden wilting when high temperatures promote the penetration of copper; but the most noticeable effect is the formation of dead, brown circular

Fig. 243.—Bordeaux spotting of apple leaf. (*After Hedrick, N. Y. (Geneva) Agr. Exp. Sta. Bul.* **287**.)

or subcircular spots 2 to 3 millimeters in diameter, or of various shapes and sizes, with many irregular in outline and apparently caused by the fusion or coalescing of smaller spots. The order of susceptibility of the leaf tissues is: (1) lower epidermis; (2) upper epidermis; (3) spongy parenchyma; and (4) palisade parenchyma, but in severe injury all are involved. If the spots are few in number no other effects may be noticed, but if numerous, the intervening tissues may turn pale green or yellow and the

leaves fall. The amount of leaf fall varies from almost none to nearly complete defoliation. A loss of one-third to one-half of the foliage was considered a fair average for New York orchards in 1905, a season very conducive to Bordeaux injury. In the most extreme type of leaf injury, the affected trees may look as though they had been scorched by fire.

The stone fruits in general, and especially the peach, are more sensitive to Bordeaux or to other copper fungicides than the apple. Spotting, burning and defoliation will be more severe with the peach than with the apple when both are subjected to the same conditions, but the peach will also show a shot holing of the foliage. In addition to the foliage and fruit injuries, a pronounced reddening of the sprayed twigs has been noted. A reduction in size of Morello and Montmorency cherries, or "small cherries," has been attributed to the use of Bordeaux. Bordeaux spraying has also been noted in certain plants as greatly increasing transpiration, and consequently increasing the injury from drought. Ginseng and Coleus have been reported as especially sensitive.

The cyaniding of Bordeaux-sprayed plants has been reported to cause increased injury from the formation of cuprous cyanide.

2. *Conditions Favoring Bordeaux Injury.*—The amount of spray injury has been exceedingly variable, even with the same varieties and with the same formula. It has also varied with localities, one region reporting heavy damage and another but little. Some of the most important features which favor or promote Bordeaux injury are as follows: (*a*) the use of excessive quantities of the mixture; (*b*) the use of too strong solutions or of those that contain an excess of copper; (*c*) mechanical injuries to the foliage, due to the presence of fungi or to the work of insects; (*d*) damp, foggy or rainy weather immediately following the application of the spray. It has been the experience of growers that the more spray they apply the greater the injury, that is, a heavy dripping spray will cause more injury than a finely divided mist spray which covers the leaves and fruit with a thin film. With the thin films, there is never so high a concentration of copper at any one point as when drops collect and thick copper deposits are formed. As a general principle, it may be stated that increase in the copper sulphate content of Bordeaux will increase the amount of injury, as will also an excess of copper over lime.

Bordeaux mixture made by using equal quantities of copper sulphate and lime or an excess of lime does not prevent injury, but the danger of injury is somewhat lessened. Insect injuries and fungous parasites of the foliage increase the injury by facilitating the penetration of the copper through the abrasions or breaks in the leaf surface. Many of the anomalies of Bordeaux injury can be explained by the weather conditions which prevail during and immediately following the spraying operations. Both field experiences and experimental tests have shown that rains and

humid cloudy weather following the use of Bordeaux greatly increase the injury. There is some evidence that synthetic fertilizers which raise the osmotic value reduce the liability to injury. Also nonbearing trees are more likely to be injured than bearing trees because of structural and osmotic differences in the leaves.

3. *Etiology.*—Bordeaux mixture is made by bringing together a solution of bluestone, or copper sulphate, and milk of lime made by slacking quicklime in water. Much has been written about the chemical composition and the physical properties of this preparation, but these features cannot be discussed at this point. If the mixture is allowed to stand, a precipitate containing all the fungicidal properties settles to the bottom. It is this precipitate which is deposited upon the plant surfaces in spraying, and under atmospheric conditions the copper hydroxide is changed into copper carbonate. Under bright sunny conditions dissolved copper does not penetrate the leaf tissues and cause injury, but under the conditions which prevail during humid cloudy weather more is brought into solution and more penetrates the plant tissues, hence the increased injury.

It has been stated by various investigators that substances secreted by the sprayed plant or by germinating spores of fungi furnish conditions for the solution of small amounts of copper. It should be noted that during bright weather transpiration is active, the stomata are frequently more or less closed and the intake of carbon dioxide is in excess of the outgo, or nearly in balance, while in humid cloudy weather transpiration is checked, the stomata are open, photosynthesis with its consumption of carbon dioxide is less active but respiration is still active, and the excess of carbon dioxide evolved passes into the water on the leaves, and the carbonated water dissolves some of the copper. The dissolved copper then diffuses through the water and into the stomatic chamber, killing the cells with which it comes in contact.

Bordeaux injury is generally slow in developing. It begins after the rains supply the requisite moisture and may continue to develop for weeks or even months. In the young growing structures, either leaves or fruits, the epidermis has not yet become cutinized, and hence penetration of the poison is possible through these unprotected walls by osmotic transfer. Bordeaux injury on the fruit results mostly from early spraying or after the blossoms have dropped, but apparently does not cause much damage later after the hairs have been shed and the stomata changed to lenticels.

4. *Susceptibility of Different Species and Varieties.*—The potato, which must be protected from late blight by the use of Bordeaux, is so tolerant of copper that little or no copper injury occurs. Peaches, apricots, Japanese plums and, to a lesser extent, Domestica plums are so sensitive to Bordeaux injury that it is seldom profitable to spray them with Bordeaux. Sweet cherries are more sensitive than the sour varieties.

Grapes also are injured by copper sprays. The apple, pear and quince are about equal in their tolerance of Bordeaux, but considerable variation in resistance is shown by the different varieties. In accordance with their relation to Bordeaux injury, apples have been grouped in the following divisions: (*a*) no injury or very slight; (*b*) slight injury; (*c*) badly injured; and (*d*) very badly injured. Important commercial varieties are found in each group, but the classification shows that the Russian varieties and crabs are generally more subject to Bordeaux injury than other varieties, although some important Russian varieties are highly resistant.

5. *Prevention.*—The danger of injury from the use of Bordeaux has led to its abandonment as an orchard spray whenever it has been possible to find a satisfactory substitute, but, for certain diseases of apples, Bordeaux is still the most satisfactory fungicide. In case Bordeaux must be used for spraying apples, it will be impossible to prevent some injury because of the influence of climatic factors over which the grower has no control. The following recommendations should be the guide so far as possible: (*a*) consider the resistance to Bordeaux injury, but select for planting varieties that are otherwise adapted to the environment; (*b*) reduce the formula to the one containing the least amount of copper sulphate that will give the desired protection, and use an excess of lime and a casein spreader; (*c*) practice moderation in spraying that is, spray with a fine mist that will cover but not drip heavily; (*d*) spray as nearly as possible in dry weather, avoiding damp, foggy or rainy periods; (*e*) remember that early sprayings are the most dangerous, and give special attention to cutting down injuries at that time.

Lime-sulphur Injury.—Lime-sulphur was first introduced into orchard practice as a substitute for Bordeaux mixture and was heralded by its advocates as a preparation which would eliminate spray injury, especially in the apple orchards, but it was soon found to cause several different types of injury.

1. *Symptoms and Effects.*—The types of injury may be: (*a*) characteristic lesions on foliage or fruit and sometimes distortion and reduction in size of leaves and leaf fall; (*b*) dropping of fruit; and (*c*) a retarding or inhibiting effect upon growth, with a resultant reduction in yield without the production of evident lesions.

The most common effect on apple foliage is a dull brown spotting or a marginal or tipburning where hanging drops of the spray have dried. A scab lesion or an insect injury usually marks the center of lesions removed from the margin, and when infections are numerous the burning may be general and severe. Even in the absence of scab, heavy drenching of the foliage may result in the burning of large areas or entire leaves. Curling or rolling of leaves with marginal injury to assume fantastic forms and reduction in size of leaves have been shown to occur in young leaves under

certain conditions, probably when the temperatures are close to the frost
line.   Severely injured leaves may be dropped and it has been shown that
this feature is aggravated by the lead arsenate which is a general accom-
paniment of lime-sulphur.   Some varieties are very sensitive to lime-
sulphur while others are rather tolerant.   The tender succulent leaves
resulting from moist, cold, sunless periods are especially sensitive to
lime-sulphur.

Peach foliage is more sensitive to lime-sulphur than apple, but the
appearance of lesions may be delayed for nearly a week after spraying
while in the apple the injury is generally evident within two days after
spraying.   On the peach, definitely outlined, usually rather pale green

Fig. 244.—Sulphur sunscald of apples.

spots appear with darker green or reddish-brown borders.   The injured
areas may drop out, thus producing a shot-hole effect.   Slight injury may
cause leaf fall, hence in severe cases defoliation may be very noticeable.
Under certain conditions, even dormant spraying with lime-sulphur has
caused injury to peach twigs and buds.   Early studies reported a practical
freedom from fruit injuries but later investigations showed that two differ-
ent types of fruit lesions may be produced: (a) a rather general russeting
somewhat similar to Bordeaux russet; and (b) circular sunscaldlike lesions
called "sulphur sunscald" on the sun-exposed cheek of the fruit.   A typi-
cal lesion is at first pale brown, later becomes darker and is flattened or
slightly depressed, and may be somewhat checked or cracked.   This type
of injury occurs mainly in the semiarid, irrigated fruit districts during
periods of high temperatures and intense sunshine.

A third injurious effect of lime-sulphur on apples is a very severe
dropping of the fruit when this fungicide is used as a spray, but this effect
is extreme only under certain climatic conditions.   In Nova Scotia, trees
sprayed with lime-sulphur (1–40) each year for seven years produced only

73.25 apples to the tree while trees of similar size sprayed with Bordeaux (3-10-50) produced an average of 341.2 apples per tree. Not only were many of the apples "sprayed off" the trees, but those which did remain were reduced in size. The yearly loss from lime-sulphur drop in Nova Scotia from 1912 to 1918 was estimated to exceed 700,000 barrels. The dropping of the fruit has also been noted in a number of other localities when lime-sulphur is used but the earlier applications omitted. Since dropping of the fruit would have been prevented by gradually accustoming the trees to lime-sulphur by the earlier applications, the behavior noted is referred to as a "sulphur shock."

The best illustration of the retarding or inhibiting effect of lime-sulphur on a crop of a different character is shown by the comparison of Bordeaux and lime-sulphur as a spray for potatoes. From tests carried out in New York during four successive seasons (1911-1914), it was shown that lime-sulphur "aggravated tipburn, dwarfed the plants, shortened the period of growth and reduced the yield." The lime-sulphur-sprayed plants died ten days to two weeks earlier than those in unsprayed rows, and the average reduction in yield per acre amounted to 28.5 bushels. Not only does lime-sulphur have this inhibiting effect upon the life processes of the potato, but it is much less valuable than Bordeaux in the control of late blight. It has been stated that lime-sulphur has a depressing effect when used as a spray for raspberries in the Puget Sound country, but this did not seem to be the case in recent Wisconsin tests. In bean plants sprayed six times with lime-sulphur (1-50) the dry weight of the sprayed plants was only 71.68 per cent of the weight of the unsprayed control. There appears to be a reduction of photosynthetic activity, and this is increased by high temperatures following the applications. Lime-sulphur has also been shown to increase the rate of respiration.

2. *Etiology.*—Lime-sulphur, which is made by boiling together lime, sulphur and water, contains calcium polysulphides (CaS_4 and CaS_5) and calcium thiosulphate (CaS_2O_3) as its most important ingredients. Both are soluble in water, but the former have been shown to be the cause of most of the injury. The other normal ingredients are practically harmless. Lime-sulphur injury appears a few days after the spray is applied, as opposed to Bordeaux injury which is generally much delayed. It is generally agreed that this behavior is due to the fact that the polysulphides remain in the soluble form but a short time, and that the intensity of the injury depends on the percentage of soluble polysulphides present.

Lime-sulphur injury will vary more or less under constant climatic conditions, being influenced by condition and susceptibility of the plants sprayed, the concentration of the mixture, the kind of arsenical employed and the time and method of application, but climatic factors are of direct

bearing, especially in the case of sulphur sunscald and the dropping of the fruit.

Experiments have shown that lime-sulphur injury is not due solely to the direct action of the soluble polysulphides, but that especially at high temperatures there is a rapid oxidation of the sulphur and the production of either sulphurous or sulphuric acid. It has also been suggested that sprayed fruit burns more than unsprayed because the spray deposit retards radiation and increases the absorption of heat. It is true that sunscald is more severe in sprayed than in unsprayed trees, and, when the temperatures are high (95°F. or above), this holds true with lime-sulphur, iron sulphide or elemental sulphur.

It is the belief that the dropping of apples and injury to other sensitive crops is due to the fact that the lime-sulphur penetrates the stomata of the leaf surfaces and acts directly on the chlorophyll bodies, causing a discoloration that can be detected by microscopic examination. This derangement of the chlorophyll apparatus inhibits or retards the photosynthetic process, and hence the young apples are "starved off the trees." This manner of action was further substantiated by certain tests: (a) lime-sulphur applied to apples alone caused no fall; (b) when sprayed on the upper side of the leaves, there was likewise no dropping; but (c) when the undersides of the leaves were sprayed, most of the apples dropped off in the same manner as in "June drop." In this connection, it should be noted that stomata are present only on the under surface of apple leaves. It is of interest to note that the foliage of the potato, grape and other plants which have stomata on the upper surface cannot be sprayed with lime-sulphur without causing serious injury. This interference with the manufacture of carbohydrates will not only explain the dropping of the fruit, but it will explain the reduced size of that which does remain and also the reduced yields in a crop like the potato, which depends on the storage of carbohydrate food. It is suggested that the difference in the susceptibility of varieties to lime-sulphur injury is due in large part to the difference in the permeability of the leaf surfaces.

It has been found that the intensity of injury varies with the amount of sunlight during May, June and July. In England, New Zealand, the Kootenay valley in British Columbia and in Nova Scotia, fruit removal by lime-sulphur seems to occur every year. When there is an average of over 250 hours of sunshine during the three critical months serious lime-sulphur drop does not occur with a normal spray program since under such conditions the chlorophyll is replaced as fast as it is decomposed.

3. *Prevention of Lime-sulphur Injury.*—The following practices are recommended for the reduction of the injury: (a) Discard lime-sulphur entirely for certain sensitive crops or for the more resistant crops under climatic conditions that are especially conducive to injury. The sub-

stitute must vary with the crop, the temperatures which prevail, the amount of sunshine and the pests to be controlled. (*b*) For tolerant crops, reduce the concentration to the lowest point which will give the desired protection. For sensitive crops like the peach use dry-mix lime-sulphur or other substitutes. (*c*) Use arsenate of lead as the arsenical, since arsenite of lime, arsenite of soda and Paris green are likely to cause serious foliage injury when mixed with lime-sulphur. Danger of injury with the combined spray of lime-sulphur and arsenate of lead is materially lessened by the use of a casein spreader, by the use of $\frac{1}{2}$ to $3\frac{1}{2}$ pounds of ferrous sulphate per 50 gallons, or in the case of the New Jersey dry-mix by the addition of 3 to 4 pounds of ferric oxide per 50 gallons. (*d*) Spray with moderation, since over-drenching is likely to cause injury, as in spraying with Bordeaux. It has been claimed that the use of the spray gun in the orchard has increased injury, presumably by covering the lower leaf surfaces to a greater extent than by the old method with mist nozzles and extension rods.

Injury from Arsenicals.—Injury from an arsenical is due to the original content of water-soluble arsenic or to interactions in a mixture which liberates free arsenic. Of the three most important

Fig. 245.—Arsenical injury to apple leaf.

arsenicals, London purple, Paris green and lead arsenate, the first is the most injurious, Paris green less dangerous and lead arsenate the safest. London purple was largely discarded in favor of Paris green, because of its large amount of soluble arsenic, while Paris green, which has shown a varying percentage of free or water-soluble arsenious oxide, has been very largely supplanted by lead arsenate, which contains but very little free arsenic.

Lead Arsenate Injury.—Spotting or burning of foliage may result from the use of lead arsenate, although this rarely causes severe injury except on the more sensitive species. Several features of interest may be noted: (1) neutral lead arsenate is generally the least injurious; (2) clear-weather spraying is safer than cloudy; (3) spraying at high temperatures is safe if the humidity is low; (4) spraying at high humidities is safe if the temperature is low. The dwarfing, shriveling and

dropping of English Morello cherries has been shown to result from pedicel injury from acid lead arsenate, when used either alone or in combination with Bordeaux, lime-sulphur or sulphur dust. Similar injury was recorded for prunes.

It has been shown that both arsenious and arsenic acid are present in commercial lead arsenate and that at low concentrations both are about equally toxic to peach foliage when compared on the basis of metallic arsenic content. At higher concentrations, however, arsenic acid is more toxic, possibly because of its greater penetrating power. For safety on tender foliage, material must be added to neutralize the free arsenic. Injury has been lessened by the addition of hydrated lime except in wet seasons on peaches, when injury may be prevented by a 4-4-1-50 of zinc sulphate, hydrated lime and lead arsenate to 50 gallons. The use of lye with lead arsenate as an apple spray has caused severe leaf spotting and black-scorch specks on the fruit from the liberation of free arsenic. Reduced acidity of oranges to 50 per cent of normal has been shown to result from spraying with acid lead arsenate, as contrasted to normal acidity when sprayed with basic lead arsenate. .

The annual addition of large quantities of lead arsenate to orchards in spraying for codling moth has led to the conclusion that both orchard trees and cover crops may be injured. Three types of arsenical injury to apple trees were first reported from Colorado: (1) *systemic poisoning,* resulting in a disturbed nutrition and growth and sometimes ending in death; (2) *corrosive* poisoning, due to localized attacks, especially at the crown, generally accompanied by internal discolorations of the wood; and (3) *bleeding,* attributed to the combined action of lime and arsenic. Similar effects have been shown to result from alkali and seepage water, and from winter injury of the blackheart and collar-rot types. More recent tests have shown that arsenic poisoning may play a part in these injuries. It still remains an open question as to the exact part played by winter conditions, alkali and soluble arsenic in the injury and death of orchard trees. It seems probable that all three factors may be operating under certain conditions, while in others only one or two of these injurious factors may be active.

Injuries from Spray-residue Removal.—During the last few years, the requirement that market fruit, especially apples and pears, must comply with the Federal standards for freedom from both arsenic and lead has focused attention upon the methods of cleaning. The various methods of spray-residue removal have been responsible for injuries: (1) burning from the liberation of free arsenic (mostly calyx burn); (2) acid burning from hydrochloric acid; (3) heat injury from the temperatures of the cleaners; and (4) increased water losses during storage resulting from the removal of some of natural wax from the skin. Arsenical

calyx burning from free arsenic was known before the days of spray-residue removal as a trouble appearing before harvest, but burning has resulted in severe form as a result of the cleaning. The other types of injuries can be reduced to a minimum by giving careful attention to the strengths of the cleaners and the temperatures employed; while arsenical injuries are now largely prevented by the use of improved machines, the proper rinsing to remove the free arsenic or the use of a neutralizing rinse.

Injury from Oil Sprays.—Before the day of Black Leaf 40, when kerosene and kerosene emulsions were the common contact insecticides, there were numerous reports of severe burning or injury, especially to vegetative structures, but the use of tobacco preparations has largely removed this danger. Oil sprays may be used: (1) as protection of trees during their dormant period; or (2) as summer applications in the control of red spider, leaf hoppers and codling moth.

Cases of severe injury have resulted from winter applications of oil when very low temperatures followed their use. The extreme injury has been the killing of whole blocks of trees, and as a result of these experiences, it is considered unsafe to spray with oil if there is danger of zero weather. Lesser degrees of injury from dormant applications include twig and bud killing. Delayed dormant applications have killed blossom buds or delayed flowering.

Before oil sprays came into such general use, the spraying of citrus fruits with distillates caused spotting and yellowing of the foliage with more or less defoliation, and spotting and dropping of fruit. The injuries from summer application of oil to both citrus and deciduous-leaved fruits include: (1) stunting, yellowing, spotting, burning and dropping of foliage; (2) dwarfing, spotting, russeting, scalding, dropping, poor coloration and delayed ripening of fruits; and (3) the formation of bark blisters, cankers or the killing of buds, twigs and bark. An extreme injury is illustrated by the complete dropping of stone fruits soon after an early oil spray.

Light oils are much less likely to cause injury than heavy oils of high viscosity. The toxic effect depends mainly on the percentages of their sulphonatable residues but, in some cases also, upon their chemical nature. The injurious effects are: (1) physical, due to insulation or sealing over of parts with consequent interference with normal gaseous exchanges; and (2) chemical from the absorption of volatile products and also from the actual penetration of nonvolatile portions. It has been shown that oil actually penetrates the stomata, occupies the intercellular spaces and may be found within the living cells of cortex, medullary rays and pith and even inside the tracheae. This penetration is explained by the oil-mass theory which postulates that the "oil touches and mixes with the oil-miscible substances concentrated in plasma membranes." (Young, 1935.)

Injuries from Seed Disinfection.—Injuries may be caused by hot-water treatments or by chemical agents, used as either liquids or dusts. The world-wide necessity for the control of seed-borne diseases of cereals has focused attention upon seed disinfection injuries.

1. *Seed Injury from Hot Water.*—The modified hot-water treatment of wheat for loose smut (10 minutes at 54°C.; allowable range, 52 to 55°C.) may cause the following injuries: (a) a reduction in the germination percentage; (b) an abnormal germination as indicated by small spindling seedlings; (c) a retardation in the rate of emergence of seedlings, and a slower growth during the seedling stage; and (d) fewer culms per plant and possibly decreased yields. The degree of injury may be illustrated by an average germination of 52.7 per cent for 33 varieties when treated by the standard hot-water method, as contrasted to 87.6 per cent germination of the untreated checks. Hand-threshed seed shows but little injury as contrasted to machine-threshed, the reduced germination being caused by seed coat cracks over the embryo and endosperm which permit the penetration of the hot water. The injury will vary, therefore, with the dryness at the time of threshing and the speed of the cylinder, and it cannot be predicted but must be determined for each individual lot of seed.

2. *Seed Injury from Copper Sulphate.*—One of the principal objections to copper sulphate which was extensively used for many years, especially for cereal smuts, has been its injurious effects on the treated seed. Wheat treated with the standard bluestone steep (1 pound to 5 gallons of water for 5 to 10 minutes) will frequently show a germination of 35 to 60 per cent as contrasted to 90 to 100 per cent germination of untreated lots. Treated seed planted in the field may show: (a) some which do not start to grow; (b) others which start to grow but are injured so much that they are not able to emerge from the soil; (c) still others which do emerge but show an abnormal development with curved deformed plumule and poor root growth; and (d) a retarded growth in others less injured. The injured seedlings seem to fall an easier prey to soil fungi.

As in the hot-water treatment, the injury from copper sulphate is caused largely by the penetration of the solution through the breaks or cracks, thus permitting it to act directly on the embryo seedling. Oats are more sensitive to bluestone injury than either wheat or barley. Since the introduction of formaldehyde, the bluestone formulas for the treatment of oats have been largely discarded, and they are used for wheat only to a limited extent, mainly in areas where there is a soil contamination. When the bluestone treatment is used, the seed injury may be eliminated, or reduced to a safe amount, by an aftertreatment or the immersion in milk of lime (1 pound to 10 gallons of water).

3. *Seed Injury from Formaldehyde.*—When formaldehyde first came into use (1893–1897), it was believed to cause only negligible reduction in

germination and, consequently, rapidly replaced bluestone. Use under diverse conditions soon showed that it also was capable of causing severe injury. The principal causes of injury are: (a) allowing the grain to dry after treatment and holding some time before seeding; and (b) seeding the treated grain in the dust so that it must remain in the soil some days before the advent of rains. This injury has been shown to be due to the formation of paraformaldehyde, a solid condensation product, or polymer, of formaldehyde, which is constantly breaking down into formaldehyde gas. In grain dried in the sac or planted in dry soil, this gas is concentrated close to the seed, penetrates the seed coats and may kill the embryo. Experiences in the dry-farming districts have shown complete failures of formaldehyde-treated wheat to germinate when planted in the dust.

The unbroken seed coats are a partial protection against formaldehyde injury but not to the same extent as with copper sulphate. Unbroken seed coats are a protection against short exposures to formaldehyde, but, with longer exposures, the gas will penetrate the unbroken seed coats and some of its initial physiological effects are a slowing down of respiration and diastatic activity. The injury is the greatest with germinations at low temperatures.

The factors which predispose to formaldehyde injury in the semiarid or dry-farming regions are: (a) the large number of broken or cracked seed generally present in the seed wheat; (b) the common prevalence of medium air humidity when seed is being prepared for seeding; and (c) the need for and the practice, in some sections, of seeding in the dust rather than waiting for a rain. Formaldehyde injury to wheat, therefore, has been most pronounced in fall-seed crops in the arid regions, but a minor factor in the humid regions and for spring seedings in the dry regions.

4. *The Prevention of Injury from Formaldehyde or Bluestone.*—The danger of seed injury can be eliminated or the hazards greatly reduced by the use of some of the newer fungicides such as copper carbonate dust, "basul" or basic copper sulphate dust, or some of the organic mercury disinfectants like Ceresan. The two copper dusts cause no seed injury, and treated seed held for a year will show a better germination than untreated seed. Ceresan-treated seed may show a loss of vitality if held long before seeding, and some organic mercury treatments (ethyl mercury chloride) have been reported to cause atypical growth, abnormal mitosis and polyploidy when used at high dosages.

If either bluestone or formaldehyde must be used, the amount of injury can be greatly reduced by observing the following precautions: (a) avoid too strong solutions or too long steeps; that is, follow standard recommendations; (b) do not attempt to dry formaldehyde-treated grain

and-hold it before seeding but treat the grain so that it can be seeded while still moist; (c) remember that wet grain will start to germinate if temperatures are favorable and may suffer injury from heating or molding previous to seeding; (d) do not plant formaldehyde-treated grain in the dust; (e) bluestone-treated seed may be planted in the dust with safety if it has been properly treated and may be dried before seeding without increase of injury (some workers even claim improved germination from complete drying); (f) protect wet grain from freezing.

In addition to the above precautions, two direct methods of reducing seed injury are available: (a) presoaking and (b) an aftertreatment.

Fig. 246.—Germination injury to wheat treated with bluestone. (*Photograph by G. L. Zundel.*)

Presoaking wheat not only eliminates or reduces seed injury due to the use of either formaldehyde or bluestone, but also increases germicidal efficiency. Seed wheat is soaked in water for 10 minutes and covered for 6 hours previous to treating. For any other seed the time of soaking should be long enough for the seeds to absorb about 30 per cent of their weight of water.

An afterbath of milk of lime (1 pound quicklime to 10 gallons of water) has long been used to prevent seed injury from bluestone and more recently has been shown to be effective in preventing formaldehyde injury. Later work has shown that the same results for formaldehyde-treated seed can be accomplished by soaking in water alone.

Injuries Due to Soil Sterilization.—Chemical preparations may also be added to the soil to kill bacteria, fungi, weeds or insects. The persistence of these chemicals in the soil or their interactions in the soil may result in injurious aftereffects upon the crop to be protected or upon following crops. For instance, if sulphur is used in large amounts to control potato scab in contaminated soils, the yield of following crops is reduced. Applications of cyanamid to the soil to kill eelworms or nematodes caused so much burning or scorching of crops planted after the treatment that it was necessary to modify the practice and the treatment has not been generally used. Carbon bisulphide has been used for soil

disinfection for nematodes or soil-infesting insects, but its use is difficult or impossible except in unoccupied areas, because of the poisonous effects upon the roots of plants. The use of sodium chlorate as a herbicide for wild morning-glory has caused injury to crops for several years following its application.

Injuries from Fumigation.—The use of fumigation as a method of distributing a fungicide or insecticide is also fraught with danger. The fumigation of potato tubers with formaldehyde for scab control resulted in so much injury that the method never came into general use. Cyanide fumigation of greenhouses for the control of white flies or other insects frequently results disastrously, since different species of plants show a varying tolerance, and seedling plants are generally more sensitive than plants of greater maturity. This makes it more difficult to be sure of a safe dosage, since, frequently, mixed cultures of varying ages must be protected. It has recently been shown that only a neutral or nearly neutral Bordeaux should be used on plants to be cyanided. The injury when nonneutral Bordeaux has been used is due to the formation of cupric cyanide. Even in cyanide fumigation of citrus trees in the open, the dosage must be very carefully adjusted to avoid injury. In this connection, the practices of anesthesia to advance the date of blossoming may be mentioned. The fumes of ether, chloroform or other anesthetic may have the desired stimulating effect, but they may also result in the death of the plant; hence the treatment requires special care.

Injuries Due to Refrigeration.—Other than freezing, injuries may result to fruit or vegetables during their storage or transport under refrigeration. Some of these are discussed in more detail under Improper Air Relations, while freezing injuries are treated in the section devoted to Low-temperature Injuries. In wrapped peaches, the browning and death of external patches, known as "ice scald," illustrate one of the difficulties encountered in refrigeration. The internal browning of the Yellow Newtown apple is due in part to holding the fruit for a prolonged period at too low a temperature. Brown heart of apples has been shown to develop under those conditions of refrigeration which were designed to prolong the storage life by retarding scald and inhibiting the action of rot-producing fungi.

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